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OF THE
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BY

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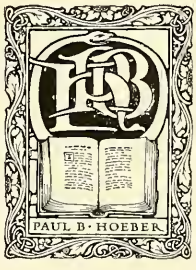

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PREFACE.

THE evolution of a technique for the prevention of external wound infection, great as was its effect in extending the scope of surgical methods of treatment in general, did not lead directly to any considerable expansion of the surgery of the chest. Though Lister had removed the obstacles to the exploration of the abdomen, the joints and the brain, and given thereby an immense impetus to the effective study of diseases in these regions, the chest remained guarded from any but the most trivial or the most desperate surgical intrusions by essential physiological conditions. It was not until Sauerbruch in 1903, at the suggestion of Mikulicz, and at about the same time Brauer, began to work on the methods of preventing collapse of the lung after free opening of the pleura, that any continuously progressive development in thoracic surgery became

possible. Though the technique of both Sauerbruch and of Brauer were to some extent and comparatively soon superseded by methods much simpler and apparently less exact, their work first decisively opened up the chest to study by the surgeon and began a development that has since then been rapid and unbroken.

So rapid has progress continued to be that the publication of a new work dealing with the present condition of knowledge in this department needs no explanation or excuse. Since the appearance of Fowler's and Godlee's admirable monograph in 1898 the subject has undergone a transformation almost complete, and since the last foreign work—that of Garré, published in 1912—a very considerable development of the whole field has to be recorded. The greatly increased simplicity of technique rendered possible by the use of intratracheal insufflation; the development of radiographic methods and the accumulation of experience derived therefrom; the working out of the special mechanical conditions which obtain in the thorax; the use of

gases within the pleura to obtain on the one hand immobilisation of the lung and on the other hand the replacement of effusions, under control by the observation of pressures and by radiography; the very large body of experience in chest injuries derived from the war—all these have contributed in their degree to rapid development and modification and to the need of a general view of the present state of the whole subject.

I have tried, while giving weight to the published work of others, to rely mainly on the data, experimental and clinical, that have come within my own actual experience; and in describing operative methods—profiting by the difficulties I have myself met with—to be closely practical.

The mechanical conditions which prevail in the chest are so distinct and special as to give to the region a pathology in some degree peculiar to itself. This mechanical factor is so important and, relatively to its importance, so little noticed, that I have not hesitated to dwell on it in a good deal of detail. Indeed it may be said that there is no part of the body where precision in

diagnosis and confidence in treatment depend more immediately upon a knowledge of how local conditions influence the processes of disease.

I have much generous help to acknowledge. Mr. Wilfred Trotter has not only read through the proofs, but has throughout given me much valuable advice in matters of arrangement and exposition.

The experience of Dr. Kincaid has been of especial value to me. He has given me opportunities to examine many interesting cases and to study his numerous radiograms. The particularly interesting radiograms Figs. 65 and 66 are from his collection. Dr. Kincaid has also read through the original manuscript and made many helpful suggestions.

Capt. Snowden, R.A.M.C., has assisted me by reading through the final proofs.

To Dr. Arthur Keith and Sir St. Clair Thompson I am greatly indebted for permission to reproduce the illustrations figured 3 and 37 in this book; and to Dr. Ironside-Bruce and

Dr. Fairbank for the radiogram Fig. 78; also to the executors of the late J. Stuart Dickey for their sanction to reproduce the anatomical drawings shown in Figs. 1, 2 and 8.

The great majority of the radiograms were taken by myself from cases under my own care. In every case illustrated, the clinical and radiographical conditions and changes have been studied side by side.

The radiograms have, with four exceptions (Figs. 36, 58, 59, 78), been taken with the plate on the front of the chest. The illustrations are viewed therefore from the same aspect as the thorax when examined radioscopically with the screen in front of the chest: *i.e.*, the left side of the chest is on the observer's right.

With the exception of Figs. 13, 27, 35, 57 and 77, the original radiograms are reproduced untouched. In Figs. 13 and 77 the ribs only have been emphasised, but in Figs. 27 and 57 the outlines of the adhesion have in each case been made more prominent.

April, 1919.

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Chapter I.

Anatomy.

An accurate knowledge of anatomy is essential to the surgeon in whatever part of the body he is operating. In the surgery of the lungs and pleura much of the work has to be done without it being possible to obtain a free exposure of the diseased part, and it is necessary that the operator should be acquainted not only with the exact position of the various structures in the chest, but in many cases also with their relative distance from each other and from the surface. The surgeon must possess in addition a knowledge of the mechanical principles in accordance with which respiration is carried on: these relate to complicated movements, and pressures which vary below and above that of the atmosphere.

The Bony and Cartilaginous Framework.

The thoracic cavity, except below and to some extent above, is surrounded by a bony and cartilaginous framework composed of the dorsal vertebræ, the 12 ribs, their costal cartilages and the sternum. This part of the skeleton, when seen from in front, has the shape of the upper two-thirds of a barrel with a triangular segment cut out of the lower part; but in horizontal section the outline bordering the thoracic cavity resembles that of a kidney, owing to the forward projection of the ridge formed by the bodies of the vertebræ and the heads of the ribs.

The Ribs.

Each rib articulates with the vertebræ at two separate points: the head, with the facets on the adjacent surfaces of the bodies of two vertebræ, and the tubercles with the transverse process of the lower of these vertebræ. At their anterior ends the upper 10 ribs are attached by means of the costal cartilages; the first 7 to the sternum, and each of the next 3 to the cartilage of the rib immediately above. The last 2 ribs have no anterior attachment.

The ribs from the 3rd to the 10th inclusive exhibit similar characteristics. The greatest curve is in the region of the angle (the ridge on the posterior aspect at the outer edge of the attachments of the erector spinæ muscles). They have an inner and outer surface and an upper and lower border: the anterior end is flat in cross-section, while the posterior part is more circular; the lower margin is prolonged downwards as a flange to form the subcostal groove which affords protection to the intercostal vessels.

The 1st rib differs from all the others : it is very short, considerably broader and flatter ; the surfaces look upwards and downwards and the borders are internal and external. The 2nd rib has characteristics which are transitional between the 1st and succeeding ribs.

The ribs, as they pass to the front of the chest, all slope in a downward direction, so that a horizontal section through the thorax would cut through three or even more ribs (Fig. 1). The intervals between the ribs increase

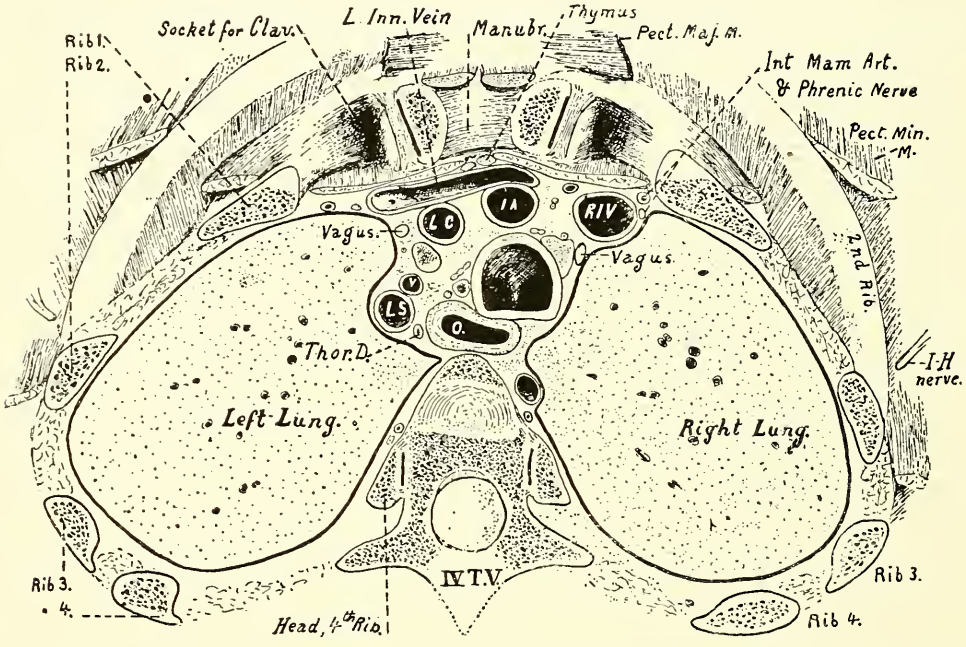

Fig. 1. Horizontal section at the level of the upper border of the sternum. The three surfaces of each lung and pleura are seen—the vertebral or vertebro-costal, the mediastinal and the costal. This section divides the 1st, 2nd, 3rd and 4th ribs. (Dickey, "Applied Anatomy of the Lungs.")

towards their anterior ends. A line drawn from the sterno-clavicular articulation to the tip of the 11th rib crosses the junctions of the ribs and cartilages, and marks also the point of widest separation of the ribs. The 1st and 2nd costal cartilages continue the line of direction of the ribs, but the 3rd to the 10th cartilages form an angle with the ribs at their juncture and run in an upward and inward direction, the interval between them lessening as they approach the sternum.

The sternum is composed of the manubrium and body, to the lower end of which is attached the xiphoid process. The manubrium and body are united by a symphysis and are normally set at an angle to each other,

the manubrium having a slightly backward inclination. The 1st costal cartilage is united by a broad surface with the manubrium, no joint, as a rule, intervening.* The 2nd to the 7th cartilages are separated from the sternum by synovial joints. Similar joints are present between the adjacent surfaces of the 6th to the 9th cartilages and often also between the 5th and 6th, and between the 9th and 10th cartilages.

The upper thoracic opening is bounded by the first dorsal vertebra, the first pair of ribs, their costal cartilages, and the manubrium sterni. Owing to the downward direction of the ribs, the upper border of the manubrium is opposite the disc between the 2nd and 3rd vertebræ,† so that the anterior part of the opening is some 5 cm. below the posterior. The relations of the first rib and cartilage require particular mention. The attachment of the scalenus anticus muscle to the roughened triangular area on the upper surface of the bone bisects the arc formed by the 1st rib and cartilage, and separates also the grooves for the subclavian artery and vein. The scalenus medius is inserted between the groove for the artery and the neck of the rib ; while opposite the contiguous borders of the scaleni, but at the outer margin of the bone, is the origin of the first digitation of the serratus magnus (serratus anterior). On the anterior aspect of the cartilage and of its junction with the rib, is the origin of the subclavius muscle, and immediately below this the attachment of the costo-coracoid ligament. The clavicle is attached to the upper border of the cartilage by the strong rhomboid ligament (lig. costo-claviculare) ; internal to this is part of the articular surface for the clavicle. The subclavian vein, passing downwards and inwards to join with the internal jugular and so form the innominate vein, is in close contact with the posterior surface of the cartilage. The left common carotid and innominate arteries are separated by the left innominate vein from the manubrium, and the vagi run behind the respective innominate veins. The internal mammary artery passes behind the subclavian vein, is crossed from without inwards by the phrenic nerve, and becomes a posterior relation of the 1st and succeeding costal cartilages. In close relation with the posterior part of the rib is the anterior primary division of the first dorsal nerve which winds up over the inner border of the bone, and, joining with the anterior primary division of the 7th cervical to form the lowest trunk of the brachial plexus, passes outwards behind the subclavian artery (Fig. 2).

The lower thoracic opening. The boundaries of this are the xiphoid process, the costal margin (*i.e.*, the 7th to 12th cartilages and the 12th rib), the first dorsal vertebra and the external arcuate ligaments (arcus lumbo-

* Sometimes a synovial joint is found occupying a part only ($\frac{1}{3}$ to $\frac{3}{4}$) of the 1st chondro-sternal juncture. This was present in 6 per cent. of the 402 cartilages I examined. It is usually, but not necessarily, bilateral.—*The British Journal of Surgery*, Vol. 1, No. 1, 1913.

† It is lower during expiration, as in Fig. 1.

costalis lateralis). The diaphragm is attached to these boundaries and the structures which leave or enter the thorax pass through openings in the muscle or its tendon.

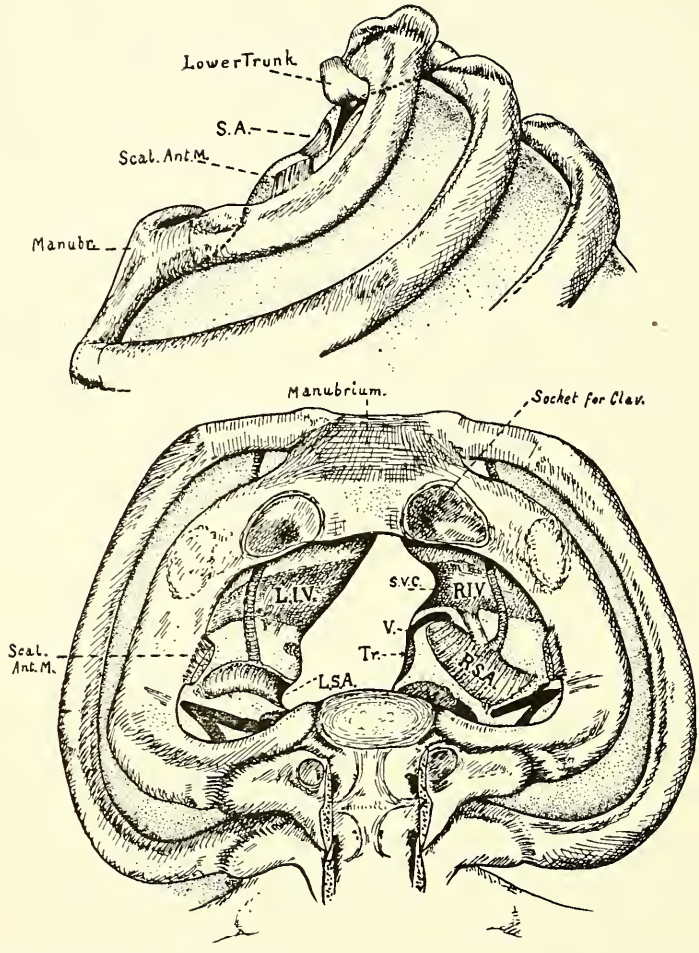

Fig. 2. Plaster casts of the pleural cavities, *in situ*: viewed from the left side and from above. S.A., impression for subclavian artery; R.I.V., L.I.V., for the innominate veins; V., groove due to right vagus nerve; Tr., impression for trachea; the V-shaped groove due to the lower trunk of the brachial plexus is seen near the neck of the first rib. The grooves due to the sympathetic and the longus colli are internal to those produced by the lower trunks, and behind those of the subclavian arteries (R.S.A. and L.S.A.) (Dickey, "Applied Anatomy of the Lungs").

The diaphragm rises into the thorax in the form of two lateral domes joined by a central tendon. The summits of the right and left domes reach as high as the upper and lower border respectively of the 5th ribs in the mammary line. On either side the domes slope steeply to the costal margin, forming a deep costo-phrenic sinus. In the mid-line, however, the central

tendon is only slightly lower than the left dome, and from its attachment to the xiphoid process, at a point on a level with the 10th dorsal vertebra, slopes gradually backwards to the 12th dorsal vertebra.

With deep inspiration and expiration the diaphragm may descend as low as the 6th interspace, or rise to the 4th interspace. The average range of movement with ordinary inspiration is 1·5 cm., but it may be as much as 7·5 cm.

Relations of Scapula and Clavicle to Thorax and Contents.

"The inner two finger-breadths of the clavicle (3 cm.) are directly in front of the cervical pleura and lung apex. No part of the thoracic wall intervenes." The 3rd, 4th, 5th and possibly the 6th finger-breadths are separated from the first intercostal space and 2nd rib "by the brachial plexus, axillary vessels and subclavius muscle" (Dickey).

When the arm is by the side, the scapula extends from the 2nd rib above to the 7th intercostal space below; this latter level corresponds with the 5th costal cartilage in front and the 6th rib in the mid-axillary line. If the arm is raised above the head, the scapula rotates and the vertebral border extends outwards, from the level of the spine of the 3rd dorsal vertebra, along the 4th rib to the 4th space in the posterior axillary line. This corresponds with the surface marking of the fissure between the upper and lower lobes.

Measurements.

The transverse diameter of the chest is greater than the antero-posterior; both increase in size from the upper opening downwards, the transverse, however, decreasing slightly beyond the 8th or 9th ribs. The average distance between the widest parts of the 4th and of the 6th pair of ribs was found by Dickey to be 24·6 cm. and 25·5 cm. respectively in the male, and 21·6 cm. and 22·56 cm. in the female.

The upper border of the manubrium sterni is opposite the disc between the 2nd and 3rd dorsal vertebræ, the sternal angle is opposite the 5th dorsal vertebra and the junction of the body, and the xiphoid process is opposite the disc between the 9th and 10th dorsal vertebræ.

The antero-posterior diameter of the upper thoracic opening is from 5 to 6 cm., while the transverse diameter is about 10·5 cm.*

The thickness of the chest wall in the mid-axillary line at the 5th intercostal space is about 0·75 cm. in a thin and 1·75 cm. in a stout person.

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* The maximum and minimum transverse diameters in a series of 200 cases examined by me were :—

Maximum diameter	Male (3)	Ages 34, 56 and 58	12 cm.
	Female	Age 37	11·75 ,,
Minimum diameter	Male	,, 36	8·6 ,,
	Female	,, 37	9 ,,

In the first space on the front of the chest the thickness varies from about 1·2 cm. to 2·8 cm.

The planes of the first pair of ribs and cartilages are oblique from behind forwards and downwards, but are on the same level. The planes of the following pairs of ribs and cartilages slope not only downwards and forwards, but also downwards and in a lateral direction, so that they form an angle with each other in the mid-vertical line, this angle becoming progressively more acute with the increase in the obliquity of the planes.

In the emphysematous or barrel-shaped chest, the obliquity of these planes in both directions is decreased ; whilst in the contracted (or so-called paralytic or phthisical) chest, the obliquity of the planes may be considerably increased. Keith has shown that the xiphisternal line is an index to the type of thorax as well as to the position of the diaphragm. This line normally crosses the 5th pair of costal cartilages : in the emphysematous chest it is at a lower level, in the contracted chest at a higher level (Fig. 3).

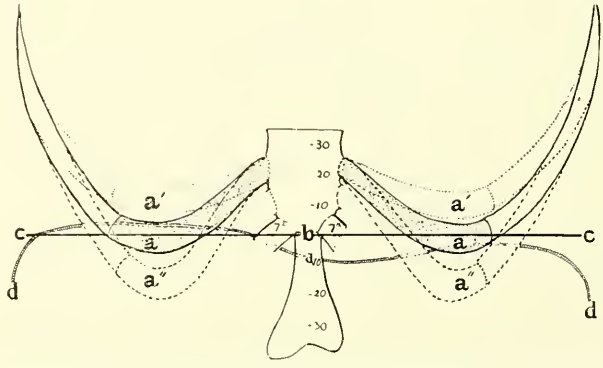

Fig. 3. The xiphisternal line and its relationship to the fifth pair of costal cartilages. a, the relationship of the line to the fifth pair of costal cartilages in a normal thorax; a', its relationship in the emphysematous thorax; a'', its relationship in the so-called "phthisical thorax"; b, xiphisternal point; c, xiphisternal line; d, position of diaphragm (average of fifty dead subjects). (Keith, *Journ. of Anatomy and Physiology*, Vol. 48.)

Changes in the Costal Cartilages and in the Manubrio-sternal Joint.

With increase in age, particularly in males, calcification and ossification changes occur in the costal cartilages so that they lose their elasticity. Ossification of the first cartilage begins usually between the ages of 30 and 40.* It is by no means infrequent to find that a fracture has occurred through some part or other of the ossified cartilage. Union may take place, but as a general rule a joint is formed between the fractured surfaces. The

* "In 125 consecutive cases, I found that slight ossification did not appear till the third decade, and marked ossification till the fourth decade. In this series, of the males over 60 years of age (34 in number), 41·2% showed slight ossification, and 58·8% marked ossification."—"A Consideration of the Influence of the First Costal Cartilage on Apical Tuberculosis."—*The British Journal of Surgery*, Vol. 1, No. 1, 1913.

ossification and calcification in the cartilages is sometimes associated with the thickening of the perichondrium ; this is particularly noticeable in the case of the first two cartilages and may be sufficient to produce, clinically, an actual prominence.

The range of movement in the manubrio-sternal joint varied in 95 cases which I examined from 0° up to 28°, the average being just under 4°. There is some evidence that the mobility of the joint decreases slightly with age.*

Anomalies of the Ribs and Sternum.

The number of ribs may be increased by the presence of a cervical or of a lumbar rib. The 12th may be excessively short and the 1st even may be incomplete, ending in a pointed cartilage attached to the sternum by a ligamentous band. The 8th costal cartilage has not infrequently a direct attachment to the sternum. The ribs, specially the 2nd and 3rd, may be bifid at their extremity : the cartilages which spring from these two portions of the bone unite, as a rule, before reaching the sternum. A bicipital rib, due to fusion of the 1st and 2nd or 2nd and 3rd ribs, is occasionally present. I have seen two cases in which the 2nd was united by a projection to the 1st rib, a synovial articulation intervening. Absence of ribs or other irregularities in development are occasionally found in association with congenital defects, such as imperfect development of the vertebral column, or Sprengel's shoulder. The sternum may be abnormally short ; the manubrium may reach to the 3rd costal cartilage. A foramen or fissure, not necessarily associated with ectopia cordis, is occasionally the result of non-union of the separate centres of ossification. Vestiges of the episternal bone of monotremata are sometimes found at the upper border of the manubrium.

The Muscles.

The diaphragm has been already discussed in connection with the lower thoracic opening, and the subclavius muscle with the anatomical relations of the 1st rib. The intercostal muscles occupy the spaces between the ribs and rib cartilages : the external, extending from the tubercles of the ribs in a downward and forward direction as far as the chondro-costal junction of the upper ribs, and continuing on to the cartilages of the lower three or four ribs. The internal intercostals are attached to the inner surface of the ribs from the chondro-sternal junction to immediately beyond the angle. The interval not occupied in front by the external intercostals, and behind by the internal intercostals, is bridged over by an aponeurotic membrane.

* Of 95 cases, 19 were between 14 and 30 years of age, and 45 between 31 and 50 years. In these two groups respectively 63·2% and 68·8% showed a movement of under 5°, whilst in the remaining 31, who were between 50 and 75 years, 84% gave a movement of less than 5°. In 19 cases there was no movement at all ; of these, nine showed complete ossification of the joint (female aged 22, three males aged 31, 32 and 34 ; one female and four males over 50).

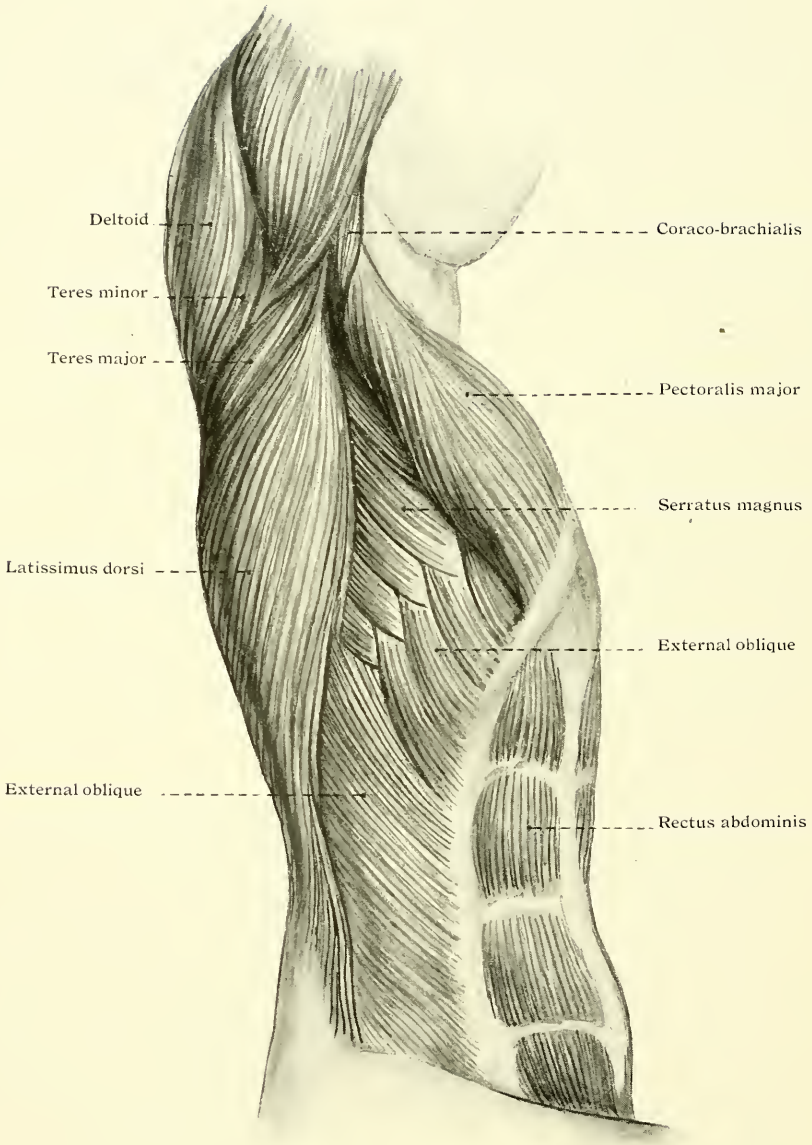

Fig. 4. The muscles covering the chest wall as seen from the antero-lateral aspect.

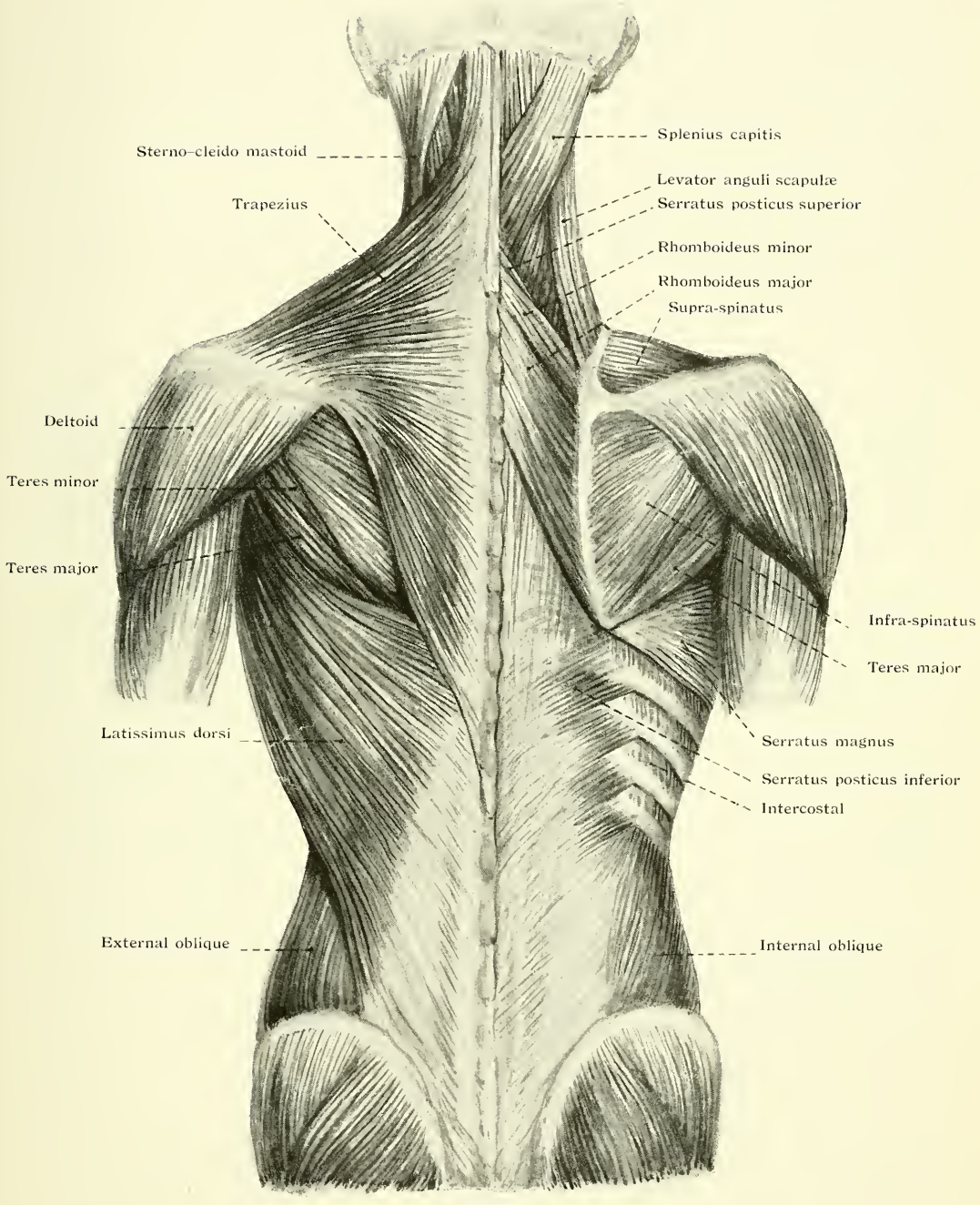

Fig. 5. The superficial and deep muscles covering the posterior surface of the chest wall.

The triangularis sterni (transversus thoracis) muscle, which is a continuation of the transversalis abdominis, extends from the xiphoid process and adjacent costal cartilages and from the lower part of the border of the sternum and is inserted into the 2nd to the 6th cartilage. The lower bundles are horizontal, while the upper ones become increasingly oblique in direction. The relations of the muscles overlying the ribs is shown in Figs. 4 and 5.

The Intercostal Vessels.

Each of the first nine intercostal spaces has an anterior and posterior blood supply. The anterior supply for the first six spaces comes from the internal mammary artery, and for the next three from the musculo-phrenic. The posterior supply for the first two spaces is the superior intercostal (intercostalis suprema), and for the last nine the intercostal branches of the aorta. The internal mammary artery rises from the first part of the subclavian and passes down the front of the chest behind the costal cartilages at a distance of about 1 cm. from the sternum in the upper spaces, but approaching nearer to that bone in the lower part of its course. At the level of the 6th intercostal space, the artery divides into its two terminal branches, the superior epigastric and the musculo-phrenic. The former enters the abdominal wall; the latter follows the costal margin. The branches from the internal mammary and the musculo-phrenic run at first deep to the internal intercostal and then perforate the muscle obliquely to inosculate with the intercostal arteries. The superior intercostal artery arises from the subclavian and reaches the first intercostal space by passing backwards and downwards between the pleura and the neck of the first rib.

The aortic intercostal arteries of the left side leave the parent trunk near the lower border of the intercostal space which they supply, and lying at first between the pleura and the internal intercostal aponeurosis they traverse the space obliquely to reach the lower border of the upper rib behind the angle. Having given off the collateral branch which runs along the lower part of the space, the arteries continue as far as the mid-axillary line between the two intercostal muscles lying in the subcostal groove. With the exception of the last two, which enter the abdominal wall, they end in the internal intercostal muscle by inosculating with the branches of the internal mammary and musculo-phrenic. On the right side the arteries must first cross over the vertebræ to reach the intercostal spaces.

The Intercostal Nerves.

The intercostal nerves are the anterior primary divisions of the first eleven thoracic nerves. They pass in front of the superior costo-transverse ligaments, the levatores costarum, and the external intercostal muscles, and are covered by the pleura. From the angle of the ribs they accompany the vessels running between the intercostal muscles. The first six are

distributed to the chest wall only, the last five are continued forwards from their respective intercostal spaces into the abdominal wall and supply the anterior abdominal muscles and skin.

While the veins and arteries are under cover of the flange of the rib, the nerves are well below the lower border of the rib lying "within the upper part of the intercostal space, but as they advance they show a tendency to occupy the middle of the space" (Piersol). The recognition of this position of the nerve is of great importance when regional anæsthetisation is attempted. A short distance in front of the angle of the rib, the nerve gives off a lateral branch which runs with it as far as the mid-axillary line and then pierces the external intercostal muscle to become cutaneous.

The Lymphatic Glands.

There are two groups of lymphatic glands in connection with the chest wall: the one is along the course of the internal mammary artery and receives lymphatics from the anterior part of the thoracic wall and the diaphragm, as well as from the abdominal parietes; the other group lies close to the heads of the ribs and receives the lymphatics from the intercostal spaces and possibly from the parietal pleura as well.

THE THORACIC CAVITY.

The Mediastinum.

The thorax is chiefly filled by two more or less symmetrical lungs; all the part lying between these is called the mediastinum. This median part is of irregular form: it extends from the upper opening of the thorax to the diaphragm, and from the vertebræ behind to the sternum in front. The principal contents of the mediastinum are the heart, the aorta, the pulmonary artery and veins, the venæ azygos, the trachea and the main bronchi, the œsophagus, the vagi and phrenic nerves, the thoracic duct, the thymus and various groups of lymphatic glands.

The upper opening is in direct communication with the neck; through it, the trachea, the œsophagus and the innominate veins enter the thorax, and the main branches of the thoracic aorta emerge; the areolar tissue of the neck continues directly into the areolar tissue of the mediastinum, no barrier intervening. The mediastinum is broadest below and in front, and makes a considerable encroachment on the left side of the chest.

The pleural cavities are not completely separated from each other by this central compartment. The pleuræ are in contact, but not in communication, behind the central portion of the sternum; while behind, between the œsophagus and aorta and vertebræ, they are in close approximation.

The Heart.

The surface markings of the heart are represented by an area roughly quadrilateral in outline. The upper boundary is a line drawn from the lower border of the 2nd left costal cartilage, $2\frac{1}{2}$ cm. from the sternum to the upper border of the 3rd right costal cartilage, 1 cm. from the sternum. The right boundary extends from this latter point to the 7th chondro-sternal junction. This line is slightly curved convex outwards, the maximum point being 4 cm. from the middle line. The left boundary extends from the left 2nd cartilage to the left 5th space, 9 cm. from the middle line. The lower boundary is a line convex downwards joining the two lowest points.

The aorta leaves the heart at a point behind the left part of the sternum opposite the lower border of the 2nd costal cartilage. The summit of the arch is opposite the mid-point of the manubrium. The right border of the ascending aorta is behind the right border of the sternum. In a radiogram the arch of the aorta is, as a rule, visible immediately to the left of the sternum, opposite the 2nd cartilage or space.

The left innominate vein crosses the summit of the arch of the aorta, joining the right innominate to form the superior vena cava, which lies to the right of the ascending part of the aorta.

The pulmonary artery extends upwards and backwards from behind the left 3rd chondro-sternal articulation to the upper border of the 2nd cartilage, where it bifurcates. It is at first in front and then to the left on a plane posterior to the ascending aorta. These two vessels are in close contact with each other and are enclosed in a tubular prolongation of the serous pericardium. Between them in front and the auricles behind is the transverse sinus, wide enough to admit the passage of a finger. The pulmonary artery is separated from the left border of the sternum and inner end of the 2nd intercostal space by the left lung and pleura. The artery ends by bifurcating into two branches which run in a horizontal direction to the right and left lung : the right pulmonary artery passing behind the ascending aorta and superior vena cava, the left pulmonary artery crossing over the descending aorta and left bronchus.

The vena azygos major (v. azygos) enters the thorax through the aortic opening. It lies on the right of the bodies of the vertebræ as far as the interval between the 5th and 4th ribs, at which point it turns forwards, crossing over, about 1 cm. above, the root of the lung, to open into the superior vena cava.

The Bronchial Vessels.

The bronchial arteries are variable in number and origin. Usually there is one on the right side arising from the first aortic intercostal, and two on the left, branches of the descending aorta. These arteries gain the

posterior surface of the bronchus, and their branches follow the ramifications of the bronchi into the lung. The blood returns partially by the bronchial veins into the systemic circulation, and partially by venules which empty into the pulmonary veins.

The Thoracic Duct.

The thoracic duct enters the thorax in company with the vena azygos major, lying between that vessel and the aorta. It lies on the dorsal vertebræ, inclining to the left, and passing behind the arch of the aorta, accompanies the left subclavian artery into the neck, where it terminates by arching outwards and forwards to open into the subclavian vein. Below the roots of the lungs the thoracic duct is in close approximation to the pleura, specially on the right side.

The Phrenic Nerves and the Vagi.

The fibres of the phrenic are derived from the fourth cervical nerve, sometimes from the third or fifth, and from the nerve to the subclavius muscle; occasionally this latter is the main source of supply. The phrenic descends over the scalenus anticus muscle, crosses its antero-internal border, and passing between the subclavian artery and vein and in front of the internal mammary artery, enters the mediastinum. The right phrenic has then an almost vertical course to the diaphragm along the right border of the superior vena cava and pericardium in front of the root of the lung. The left phrenic crosses in front of the left vagus from without inwards, passes over the arch of the aorta and follows the contours of the pericardium to the diaphragm. The phrenic nerves contain sensory as well as motor fibres.

The right vagus enters the thorax, passing in front of the first part of the subclavian artery and behind the right innominate vein; it then runs along the side of the trachea, passes behind the vena azygos major and reaches the posterior aspect of the hilum. The left vagus gains the root of the left lung by crossing over the arch of the aorta. On either side the nerve breaks up on the posterior surface of the hilum and, joining with filaments from the sympathetic, forms the posterior pulmonary plexus, whence branches pass into the lung in company with the bronchi. The anterior pulmonary plexus is formed by a few branches from the vagus which are given off at the upper border of the hilum and are joined by filaments from the sympathetic. At the lower border of the roots of the lung, the vagi are continued from the posterior pulmonary plexus on to the œsophagus, the right running along the posterior and the left along the anterior surface of that organ.

The Trachea and Bronchi.

The trachea extends from the cricoid cartilage above, to the lower border of the manubrium sterni, opposite the lower border of the 4th or

the upper border of the 5th dorsal vertebra. It is from 10 to 12 cm. long and has from 16 to 20 ∩-shaped bars of cartilage in the anterior and lateral walls. "It is separated from the skin by a distance of $1\frac{3}{4}$ inches ($4\frac{1}{2}$ cm.) or more at the level of the top of the sternum in a well-grown man, and it is usually from $2\frac{1}{2}$ to 3 inches (6 to $7\frac{1}{2}$ cm.) from the surface at its bifurcation" (Godlee). The trachea, on entering the thorax, occupies a median position between the innominate artery and the left carotid. It is crossed by the left innominate vein and the arch of the aorta, the deep cardiac plexus lying between it and the latter structure. The right vagus and recurrent laryngeal (n. recurrens) are in relation to the right side of the trachea, and the left recurrent to the left side. Behind, the trachea is separated from the bodies of the vertebræ by the œsophagus.

The bifurcation of the trachea lies behind the lower border of the arch of the aorta. The right bronchus, which is the larger, forms a much more obtuse angle with the trachea than does the left, and continues more nearly the direction of the trachea. On either side, the bronchus entering the hilum at the upper and posterior part is continued downwards to the base of the lung, running close to its internal aspect, and ends near the inferior surface of the lower lobe, each bronchus giving off large branches to the several lobes.

The right and left bronchi are crossed by the corresponding main branches of the pulmonary artery at a point about 3 cm. below the bifurcation of the trachea.

The right bronchus gives off its first main branch at a distance of 2 cm. from the trachea, and before, therefore, it has been crossed by the pulmonary artery. This branch which supplies the upper lobe is known as the eparterial bronchus and divides into an ascending, posterior, and anterior branch which go to the respective parts of the lobe. Below the level of the pulmonary artery, at a point 4 cm. distant from the bifurcation of the trachea, the bronchus gives off, on the anterior aspect, a large branch which supplies the middle lobe. The next branch arises from the posterior aspect at a distance of $5\frac{1}{2}$ cm. from the bifurcation of the trachea, and passing outwards and backwards divides into branches which supply the upper and posterior aspects of the lower lobe. Below this again a series of branches radiate in a downward direction from the main stem and are distributed to the basal portions of the lower lobe (Fig. 6).

On the left side the first branch from the bronchus comes off below the level of the pulmonary artery, at a distance of 4 cm. from the bifurcation of the trachea. It divides at once, the upper division breaking up into an ascending, posterior, and anterior branch, as in the case of the eparterial bronchus; while the lower, corresponding with the branch which supplies the middle lobe on the left side, passes in a downward and forward direction to the lower part of the upper lobe. The distribution of the branches to the lower lobe is similar to that on the right side.

The trachea, bronchi and bronchioles are lined by ciliated columnar epithelium, the infundibula by cubical epithelium and the alveoli by pavement epithelium. Elastic tissue is present in the walls of the bronchi and of the air cells.

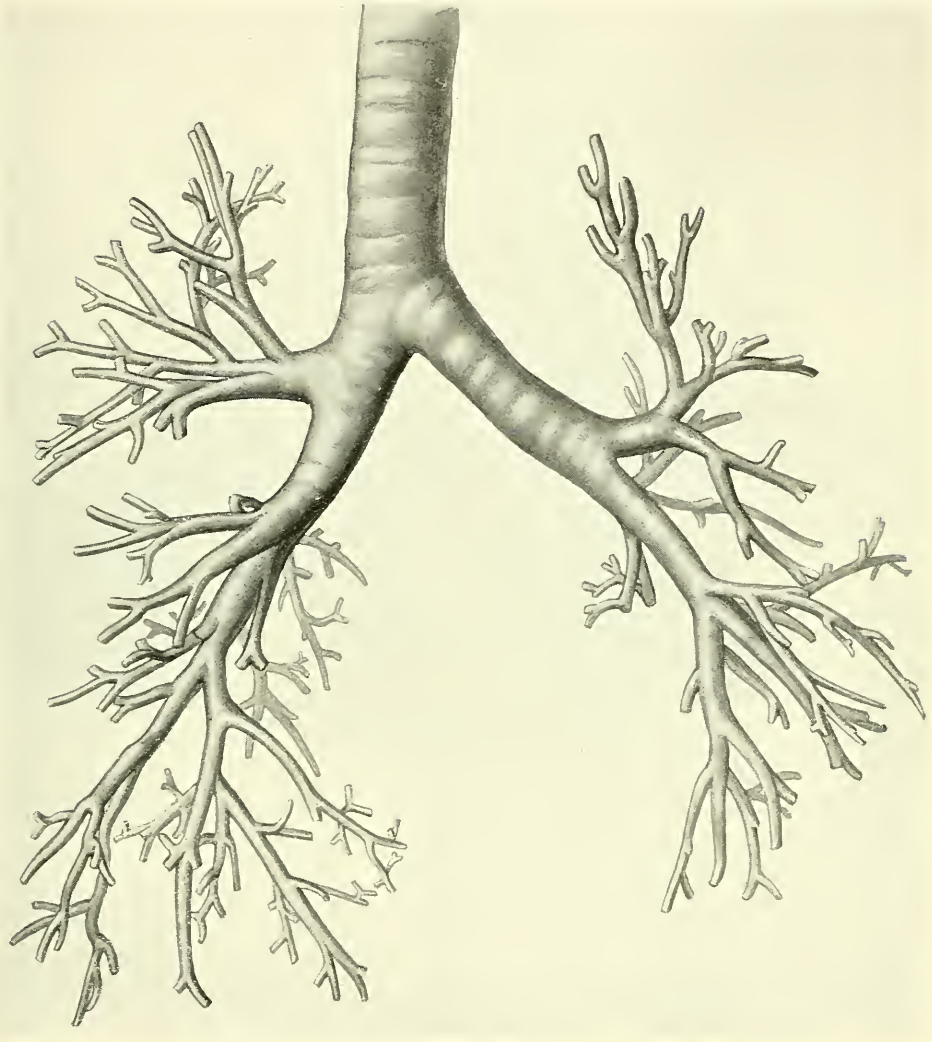

Fig. 6. Cast of the bronchial tree (made by the author). The smaller bronchi have been omitted for the sake of clearness. The bronchus for the middle lobe arises from the anterior surface of the main bronchus in close proximity to the highest branch for the lower lobe.

"The bronchial muscles are supplied by powerful broncho-dilator nerves which are sympathetic in origin, and by broncho-constrictor fibres through the vagus" (Dixon and Ransom, *Jour. of Physiol.*, 1912). "The bronchial musculature, by diminishing or increasing the access to the

infundibula in various parts of the lung, may regulate the distribution of the indrawn air throughout the lung. By regulating the intra-alveolar pressure it may also influence the distribution of blood throughout the lung, and take the part of the vaso-motor mechanism which has not been proved to exist in the lung." (Keith, "The Mechanism of Respiration in Man. Further advances in physiology," page 187.)

The Pleural Membranes.

Each of the lateral cavities of the thorax is lined by a membrane, which is reflected from the mediastinal surface along the hilum and broad ligament on to the lung. The part lining the cavity and that investing the lung are known as the parietal and visceral pleura respectively. The pleural membrane consists of fibrous tissue covered by endothelial cells.

The Parietal Pleura.

According as it covers the inner surfaces of the ribs, the vertebræ, the mediastinum, the diaphragm, or extends upwards into the upper thoracic opening, it is known as the costal, costo-vertebral, mediastinal, diaphragmatic and cervical pleura respectively.

The lines of reflection of the pleura off the lung and mediastinum and off the diaphragm mark the limits of the pleural cavity. These lines of reflection and the relations of the cervical pleura need further description. The anterior line of reflection extends from behind the sterno-clavicular articulation downwards and inwards to the mid-point behind the sternal angle, where the pleural membranes of the two sides come into apposition. The pleuræ continue in apposition inclining slightly to the left till opposite the 4th chondro-sternal junction, where the line of reflection of the left side turns outwards to the margin of the sternum and then again vertically downwards to the 6th cartilage. The right line of reflection turns outwards opposite the junction of the 6th or 7th cartilage with the sternum.

The inferior or diaphragmatic line of reflection is a continuation of the anterior line and is practically the same on the two sides. The reflection takes place along the 7th costal cartilage, crosses the 8th chondro-sternal junction immediately external to the mammary line, and reaches the 10th rib, or possibly the 10th space, in the mid-axillary line. This is the lowest border of the costo-phrenic pleural sinus. From this point it proceeds backwards and slightly upwards across the 11th and 12th ribs and reaches the spine, on the left side immediately below the head of the rib, and on the right side at a slightly higher level. (Figs. 7 and 9.)

The relations of the postero-internal line of reflection, where the pleura passes from off the vertebræ on to the mediastinum, are of special importance. Below the roots of the lung and behind the broad ligament on the right side,

the pleura extends inwards behind the mediastinum and the œsophagus, and in front of the 8th and 9th vertebræ, the vena azygos major and possibly even the aorta. This is the vertebro-mediastinal pleural sinus. On the left side also there may be a slight internal protrusion of the pleura. "This

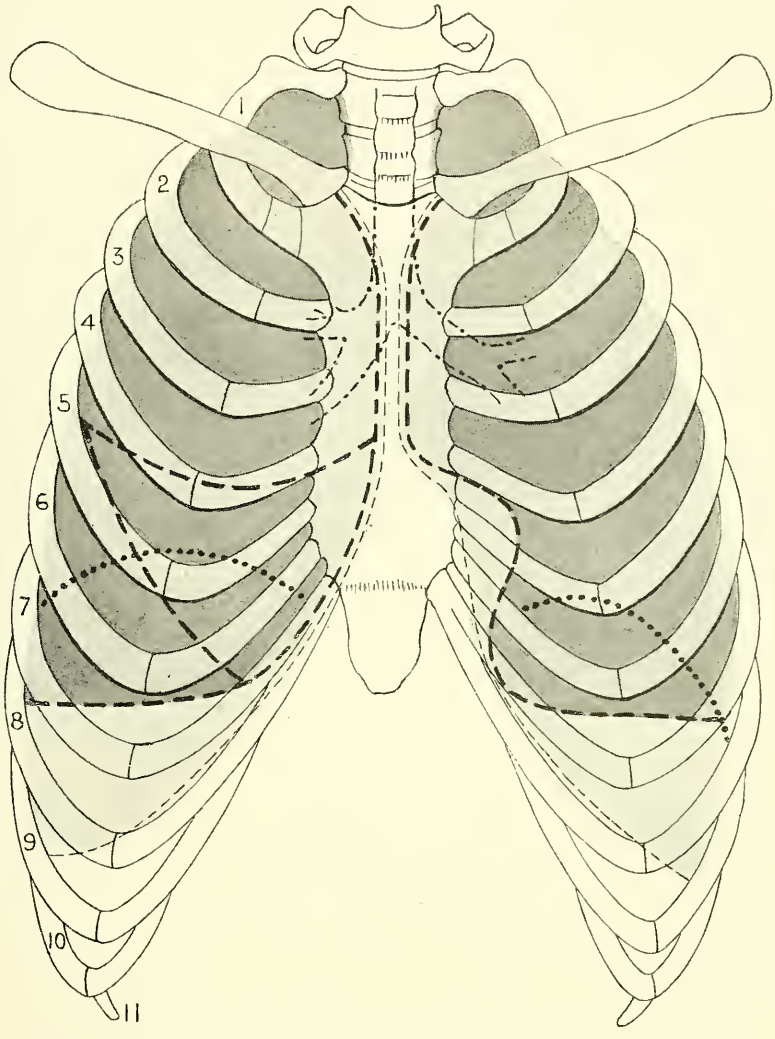

Fig. 7. Front view of the skeletal thorax, showing the relationship to the bony parts of the lung (dark shading), of the pleura (light shading), and of the trachea and primary bronchi. The position of the diaphragm and of the fissures of the lung is also indicated.

fact seems in part to explain not only the lateral mobility of the mediastinum, but possibly also the occasional extension of morbid processes from one pleura to another" (Godlee). It is at this point also that the pleura of both sides comes into close relation with the thoracic duct. Above the root of

the lung, the line of reflection is in relation on the right side with the trachea and innominate vessels, the superior vena cava, the vena azygos major and the vagus and phrenic nerves. On the left side it is in contact with the œsophagus, the arch of the aorta, the subclavian artery, the left innominate vein and with the vagus and phrenic nerves.

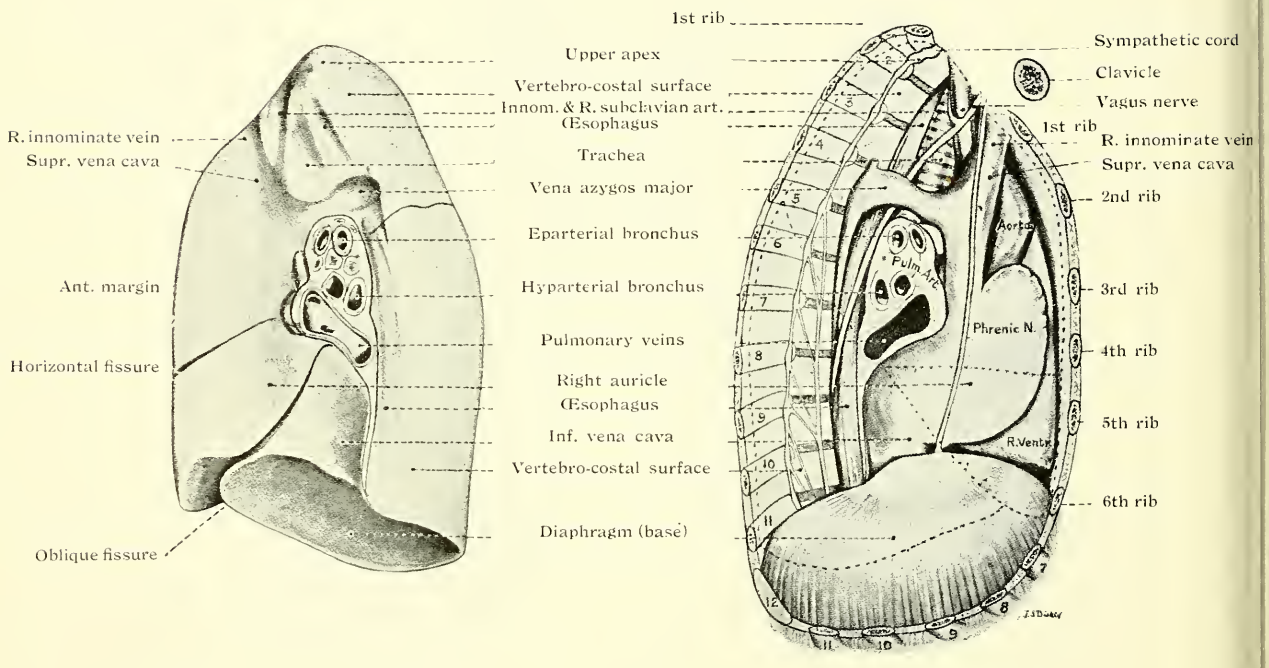

Fig. 8. View from the medial aspect of the right lung of an adult. The parietal pleura and the pericardium are represented as removed in the diagram, in order to expose the underlying structures. The club-shaped outline of the root of the lung and the broad ligament are shown bounded by a dark line indicating the pleura. The right pulmonary artery has divided into an upper and a lower branch, which lie in front of the eparterial and hyparterial bronchus respectively. The two pulmonary veins have blended. (Dickey, "Applied Anatomy of the Lungs.")

At the root of the lung the parietal pleura is reflected on to the hilum and becomes continuous with the visceral pleura. Below this level, the pleuræ are continuous along the mesentery-like ligament stretching from the mediastinum to the posterior and internal aspect of the lung. This band of pleura, the broad ligament, is attached above to the hilum; below, it ends with a free border close to the diaphragm.

The cervical pleura extends upwards into the upper thoracic opening. The highest point is on a level with the lower border of the neck of the first rib behind, *i.e.*, about 4 to 5 cm. above the manubrium. The relations of the cervical pleura are as follows* :—In the middle line are the thymus gland, left innominate vein, trachea, œsophagus, left recurrent laryngeal nerve and the longus colli muscles. On the right of the trachea are the innominate artery and right innominate vein, with the right vagus nerve between them. On the left, the left carotid and subclavian arteries, the left vagus nerve and the thoracic duct. On either side are the inferior cervical ganglion of the sympathetic and the first dorsal nerve, the superior intercostal artery intervening between these two structures. The internal mammary artery and the phrenic nerve are in front of the pleura together with the various lymphatic glands.

The cervical pleura needs strong support owing to the constantly varying intrathoracic pressures. In addition to the osseous ring of the upper opening and to the scalenus anticus and medius muscles, there is the support afforded by the scalenus pleuralis. This muscle terminates in the expansion which is attached to the cervical pleura and is known as Sibson's fascia. Further support is given by certain ligamentary bands which pass to the pleura from the deep fascia and from the neck of the first rib.

The parietal pleura is supplied with sensory fibres from the intercostal nerves. The mesial portion of the diaphragmatic pleura obtains its sensory fibres in part, and the pericardial pleura entirely, from the phrenic nerves. The visceral pleura is insensitive.

The Lungs.

These organs fill the greater part of the pleural cavities, but do not extend to the extreme limits of the deeper pleural sinuses. The portions of the pleural cavities not occupied by lung under normal conditions are three in number :—

(1) Between the 4th and 5th costal cartilages on the left side. The pericardium is in this region therefore separated from the chest wall by two layers of pleural membrane only.

(2) The costo-phrenic pleural sinus. The lower border of the lung extends down as far as the 6th costal cartilage at the border of the sternum, the 7th space in the mid-clavicular line, the 8th rib in the mid-axillary line and the 10th rib in the scapular line. The level is slightly higher on the right than on the left side.

(3) The vertebro-mediastinal pleural sinus.

When the lung is markedly emphysematous, these spaces may be encroached on, but the lung probably never reaches to the bottom of the costo-phrenic sinus.

* This account and the description of the supports of the pleura are taken from Dickey.

Lobes and Fissures of the Lung.

On the left side the lung is divided into two lobes by the oblique fissure which extends outwards from the region of the hilum. The surface marking

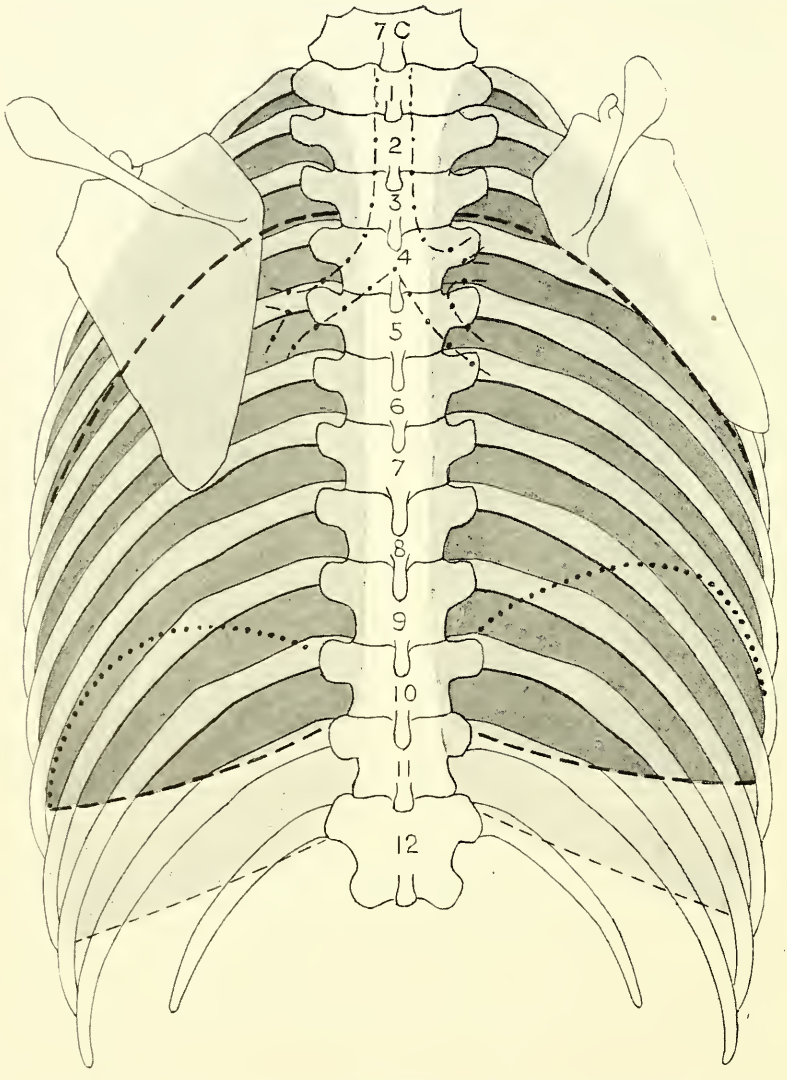

Fig. 9. Back view of the skeletal thorax, showing the relationship to the bony parts of the lung (dark shading), of the pleura (light shading), and of the trachea and primary bronchi. The position of the diaphragm and of the fissures of the lung is also indicated. On the right side the scapula is shown in the position assumed when the arm is raised above the head. The vertebral border then corresponds with the posterior part of the oblique fissure.

of this fissure is a line which starts from a point at the outer border of the vertebra opposite the 3rd dorsal spine, ascends slightly to the lower border

of the 3rd rib in the scapular line, and then descends obliquely across the side of the chest, crossing the 5th rib or space in the mid-axillary line to terminate opposite the 6th rib or space at or internal to the mid-clavicular line. On the right side the lower lobe is separated from the upper and middle lobes by the oblique fissure which corresponds in position to that on the left. The upper and middle lobes are separated by a second fissure which branches off from the oblique one opposite the 5th rib between the posterior and mid-axillary lines, and runs horizontally forwards to behind the 4th chondro-sternal juncture at the anterior border of the lung. The part of the lung on the left side which is in contact with the diaphragm consists almost entirely of the basal surface of the lower lobe, with in front, near the middle, a small strip of the inferior border of the upper lobe. On the right side the upper lobe does not come into relation with the diaphragm, but the middle lobe is in contact with this muscle in front and the lower lobe is in contact behind, and to the outer side.

The Hilum.

Each root of the lung contains the main bronchi, the pulmonary artery, and the two pulmonary veins, together with the bronchial vessels, the lymphatic vessels and glands and the pulmonary plexuses. The relation of the bronchi to the pulmonary vessels differs on the two sides. The bronchus is above and behind the artery on the right, below and behind it on the left. On both sides, the one pulmonary vein is the most inferior and the other the most anterior of the important structures of the hilum.

The right pulmonary artery divides into two branches, the one supplying the upper lobe and the other the middle and lower lobes. On the left side also the artery divides into two, one branch for each lobe. The course of the other structures has already been described.

The root of the right lung is situated opposite the spinous processes of the 4th and 5th dorsal vertebræ, that of the left lung is at a slightly lower level.

Lymphatics.

There is a chain of glands running along the posterior part of the mediastinum closely connected with the descending aorta and œsophagus. In the anterior part of the mediastinum there is a group of glands lying between the manubrium in front and the innominate veins and aorta behind. The bronchial glands consist of a group situated at the angle of divergence of the bronchi, and of chains of smaller glands lying in contact with the bronchi and their main branches in the hilum.

The lymphatics of the lung consist of a superficial or subpleural and of a deep set which accompanies the bronchi. These two sets communicate at the hilum

The lymphatics of the parietal pleura sometimes communicate with those of the chest wall.

MOVEMENTS OF THE THORAX AND LUNGS.

The description of the movements of the diaphragm, ribs and lungs is taken directly from Keith, "The Mechanism of Respiration in Man."

Movements of the Diaphragm.

The diaphragm consists of two parts, the spinal or crural and the sterno-costal. Contraction of the fibres of the spinal part produces an elongation of the thorax in a vertical direction, while contraction of the sterno-costal part increases the antero-posterior diameter of the lower part of the thorax. "The resultant movement of the diaphragm is one in a downward and forward direction." The action of the diaphragm is closely related with the action of the interchondral parts of the internal intercostal muscles. During inspiration, these muscles and the anterior digitations of the diaphragm raise the cartilages "into a more horizontal position, increasing the transverse diameter of the thorax and the size of the subcostal angle. The cartilages are depressed and the subcostal angle decreased by the upper part of the transversalis and triangularis sterni." Keith regards a free movement of the subcostal angle as an index of a free action of the diaphragm.

Movements of the Ribs.

The 1st rib and the manubrium move as one piece between the costo-vertebral articulations and the manubrio-sternal joint. The elasticity of the cartilages allows only a very small amount of play between the ribs and sternum. During inspiration, the manubrium and the first pair of ribs are raised and the antero-posterior diameter of the chest is increased.

The movements of the 2nd to the 5th ribs are on an axis which corresponds to their necks. Their action is therefore to raise the sternum upwards and forwards and so to increase the antero-posterior but not the transverse diameter of the chest. The greater the mobility of the manubrio-sternal articulation, the greater is the freedom of movement of the first and succeeding four pairs of ribs.

The 6th to the 10th ribs rotate round a sterno-spinal axis and they, in conjunction with the diaphragm, increase the transverse and antero-posterior diameter of the lower part of the thorax and the vertical diameter of the whole chest.

Movements of the Lungs.

The extensibility of the lung is least near the root and increases, gradually at first and then more rapidly, as the surface is approached and the fine bronchi and vessels cease to offer marked resistance to expanding forces. The expansion of the lung is also influenced by the surroundings. During

normal respiration there is but little movement of the structures to which the vertebral, mediastinal and apical surfaces of the lung are opposed; the expansion of these portions is consequently mainly indirect. The costal and diaphragmatic parts are however in contact with freely-moving surfaces which exert a direct influence on them. The roots are intimately connected with the heart, which moves downwards with the diaphragm and forwards with the sternum.

The lung is divided by the longitudinal fissure into an upper and anterior and a lower and posterior portion. The upper is expanded by the mechanism of the upper ribs, the lower by that of the lower ribs and diaphragm. As has been stated above, the enlargement of the upper part of the chest during inspiration is by a costo-sternal mechanism, increasing the antero-posterior diameter of the thorax. The upper lobe is expanded by this mechanism and probably does not alter its position relative to the chest wall. The lower part of the chest is enlarged in the lateral and vertical as well as in the antero-posterior direction. The expansion of the lower lobe is therefore more complex: with the descent of the diaphragm and the raising of the ribs, the surface of the lobe moves downward over the chest wall and occupies a relatively lower position during the inspiratory than during the expiratory phase.

The indirect expansion of the more fixed parts of the lung surfaces is due mainly to the increase in the vertical diameter of the chest by the descent of the diaphragm, and the increase in the antero-posterior diameter by the combined action of the diaphragm and ribs on the sternum.

Chapter II.

INTRAPLEURAL PRESSURES.

The surface of the lung is everywhere in close contact with the parietal pleura, a very thin film of fluid alone intervening. In the potential space between these two pleural surfaces there is a negative pressure which is sufficient to overcome the traction of the elastic tissue of the lung even at the moment of the maximum expansion of the chest. This negative pressure or partial vacuum is the main factor in maintaining the lung in a condition of expansion : the capillary attraction between the two surfaces of the pleural membranes is a contributory cause. The intrapleural negative pressure is equal, in a normal subject, to about 6 mm. of mercury, and shows only a very slight excursion with inspiration and expiration. The excursion may be as much as 1 mm. of mercury, during quiet respiration, but is as a rule less. It is surprisingly small in comparison with the increase in size of the pleural cavity during inspiration, but this is accounted for by the flexibility of the elastic tissue, by the freedom of entry of atmospheric air into all parts of the lung, and by the fact that the intrapleural space is a potential one only, the apposition of the pleural membranes being aided by the capillary attraction. With deep inspiration or forced expiration the negative pressure may be increased or diminished a further 1 mm. of mercury. So soon as the intrapleural space is made real by the introduction of a gas, the excursions during normal respirations are immediately increased up to 3, 4, or even 5 mm. of mercury, but rarely more except under exceptional circumstances to be mentioned later.

The negative pressure is diminished in certain cases, such as emphysema, and is increased in others, such as fibrosis of the lung and pneumonia. Changes in the lung appear to have more influence on the pressure and on the range of the excursions than do changes in the movements of the chest wall and diaphragm. There is little, if any, difference in the range of the excursions during respiration in the male and female. Patients suffering from phthisis with a retracted chest and a badly moving diaphragm are usually found to have a very limited excursion ; this is probably due to the fibrosis of the lung, as it is not necessarily found in early cases of phthisis where the fibrosis is less marked, but in which the diaphragm is moving little or not at all. In one case, I have the records before and after section of the phrenic nerve resulting in paralysis of one half of the diaphragm and there is practically no difference either in the degree of the intrapleural negative pressure or in the excursions during quiet respiration. In this patient the costal movements

were good. A healthy lung gives a wider excursion than a fibrosed lung, since in the latter the tissues are capable of less expansion and therefore of less recoil. The associated contraction of the chest doubtless contributes to the diminution of the excursion, but is probably a subsidiary factor.

The presence of a partial pneumothorax, as has already been said, allows of increase in the variations of intrapleural pressure, and if free expansion of the lung is impeded by intrapulmonary fibrosis, the excursions tend to increase with decrease of the negative pressure. When the pressure is positive during both inspiration and expiration, the excursion is definitely greater provided there are no adhesions (a high pressure in a small pneumothorax limited by adhesions is accompanied by very slight, if any, variations). In one patient, in whom spontaneous pneumothorax developed associated with dyspnœa, I found a positive pressure equal to 24 mm. of mercury during expiration, and equal to 1 mm. of mercury during inspiration.

A rapid decrease or increase of intrapleural pressure causes distress to the patient : it may produce grave symptoms and even death. On the other hand, if the change be brought about slowly, a very high negative or a considerable positive pressure can be tolerated with few or no symptoms at all. A rapidly accumulating pleural effusion will cause pain, shortness of breath and cardiac embarrassment with a comparatively small bulk of fluid ; whereas with an effusion which accumulates slowly, the only symptoms may be a feeling of tightness in the chest and shortness of breath on exertion, even though one pleural cavity is filled with fluid and the heart is considerably displaced. The same applies to positive pressure due to gases ; a spontaneous pneumothorax produces far more serious symptoms than a pneumothorax of the same intensity brought about gradually.

A patient can tolerate a very much higher negative than positive pressure. During ordinary aspiration, a feeling of tightness is, as a rule, complained of when the negative pressure is equal to about 8 or 10 mm. of mercury ; while a rise equal to another 2 to 4 mm. of mercury usually causes severe pain and coughing. If the aspiration is continued beyond this point, œdema of the lungs and death may ensue. Toleration, however, is easily acquired, and I have the records of one patient in whom a negative pressure equal to 54 mm. of mercury during normal inspiration could be produced without any discomfort whatever. A big inspiration at this stage raised the negative pressure to considerably over 60 mm. of mercury, which was the limit it was possible to record on my manometer.

It is obvious, from the above, that when the intrapleural pressure is under control, considerable variations can be produced not only without harm, but also without discomfort to the patient. No operation in the closed chest which affects the intrapleural pressure should be done therefore without there being some means of knowing every variation of the pressure, and of being able to regulate this pressure as required. Control can be obtained by means of the apparatus shown in Fig. 14 and described on

page 50, or that shown in Fig. 50 and described on page 165. Without such control the patient will almost certainly suffer discomfort and probably pain, he may be acutely distressed, and there is even the extreme chance that he may die. Further, absence of control necessarily implies a considerable ignorance of what changes are taking place in the chest at any moment, and consequently, the results obtainable are limited in scope and are in some measure speculative in character.

Open Pneumothorax.

If a large opening is made into one side of the chest, the pressure in that pleural cavity becomes zero, that is to say, the air on the outer surface of the lung is at atmospheric pressure and equal to that in the main bronchi. Since the pressure outside and inside the lung is the same, the lung collapses as the result of the traction of its elastic tissue. Such a condition of open pneumothorax is one of great danger to the patient and the seriousness is due, not to the collapse of the lung, but to the varying pressures in the opposite side of the chest and to the effect of this on the mediastinum. When the open pneumothorax is made, the negative pressure on the closed side pulls on the mediastinum, which is normally kept in a state of equilibrium by the equal tension on either side of it. During inspiration, the mediastinum is drawn still further over to the closed side of the chest and necessarily interferes with the expansion of that lung, diminishing still further the respiratory capacity of the patient. But even this diminution is not sufficient to endanger the patient's life so long as there is no undue exertion, such as restlessness or coughing. Just as during inspiration the mediastinum is drawn over to the closed side, so during expiration does it return to its former position and beyond. There is therefore a constant to and fro flapping of the mediastinum and of its contents which is increased during laboured breathing and becomes greatly exaggerated by coughing. This constant movement has a deleterious effect upon the heart, and produces a state of shock which, if allowed to continue, ends in the death of the patient from cardiac failure.

The conditions described above obtain in all cases of open pneumothorax except those in which the lung and mediastinum are fixed, or their movements are limited by thickening of the pleura and adhesions (as in most cases of empyema); and except in those also in which the opening through the chest wall is less in size than the smallest part of the upper respiratory tract, *i.e.*, the glottis. If a cannula is passed through the chest wall, there will be gradual collapse of the lung on that side. As, however, the passage of air through the glottis is freer than that through the cannula, there will continue to be slight variations in the volume of the collapsed lung, whilst the to and fro movements of the mediastinum will be insignificant in degree and the patient's life is consequently not endangered.

It is clear, therefore, that an uncompensated open pneumothorax is dangerous so long only as the lung or mediastinum is in no way fixed by adhesions, and so long as the opening in the chest is larger than that of the glottis. Even in these circumstances the degree of shock produced is not serious if the mediastinal flapping does not continue for more than a few minutes.*

Intrapleural operations with an uncompensated pneumothorax may be divided into three groups :

(1) Those cases in which adhesions are present and serve to fix the lung or mediastinum, such as in empyemata and in some cases of gangrene and abscess.

(2) Those cases in which, though adhesions are not present, no extensive and continuous inspection of the interior of the chest or dissection of tissues within the pleural cavity is necessary, such as in cases of gunshot wounds, of gangrene and abscess, or of hydatids. In this group the only intrapleural manipulation required is the palpation of the lung for the localisation of the lesion ; the affected area is then drawn up to the opening in the chest wall, and is fixed there either as a temporary or permanent measure during the subsequent stages of the operation.

(3) Those cases in which intrapleural dissection is an essential part of the operation, such as ligature of the branch of the pulmonary artery and pneumectomy in the absence of adhesions.

It has already been shown that the pneumothorax in the cases mentioned in group (1) is not dangerous, and that no special precautions are necessary. During operations which come under group (2), the dangers of an uncompensated pneumothorax can be greatly minimised if, at the same time as the hand is inserted into the pleural cavity to localise the lesion, a wet towel is placed over the incision through the chest wall and round the surgeon's forearm. When the lesion has been found, that part of the lung must be brought up to the wound and held there, or stitched to the margins of it. In doing this the traction necessarily exerted on the lung and hilum prevents movements of the mediastinum.†

The operations in group (3) do not lend themselves to such simple measures, since the opening into the chest is necessarily a very large one and fixation of the lung is not possible. More elaborate methods must be adopted to overcome the dangers of the mediastinal flapping. These entail the use of more or less complicated and special apparatus, such as are detailed below.

* The danger of secondary infection through the opening is, of course, not under consideration in this chapter.

† In these cases, although the uncompensated pneumothorax does not necessitate *per se* the use of special apparatus, yet, as is shown later, there are other reasons which make such apparatus advisable.

It may be said in passing, however, that in cases of emergency or where no form of apparatus is available, the mediastinal flapping may be to some extent controlled, if that part of the pleural cavity the free exposure of which is not necessary to the operation is lightly packed with gauze or towels.

APPARATUS FOR THE CONTROL OF OPEN PNEUMOTHORAX.

In the normal state the pressure on the surface of the lung is less than that in the bronchi. An open pneumothorax renders both pressures approximately equal. In order to compensate the pneumothorax, it is necessary either to diminish the pressure of the air on the surface of the lung or to increase the pressure in the bronchi. The numerous forms of apparatus which have been devised to this end work on one or other of these two principles. Those which effect compensation by reducing the external pressure are termed negative pressure or hypo-atmospheric apparatus, while those which increase the intrabronchial pressure are called positive pressure or hyper-atmospheric apparatus.

Sauerbruch, at the instigation of Mickulicz, was the first person seriously to study this problem. He invented both a hypo-atmospheric (Unterdruck) chamber and a hyper-atmospheric apparatus (Überdruck). The latter he abandoned for the former, finding that this reproduced more closely the normal physiological conditions.

The Hypo-atmospheric Chamber.

The principles of this chamber are that the chest is opened in an atmosphere which is at a negative pressure equal to about 8 mm. of mercury, whilst the patient breathes air at normal pressure. The chamber is in reality a small operating theatre, which has double doors with an airlock between them. The air in the whole chamber is maintained at the reduced pressure by a motor and pump. The surgeon and his assistants work inside the room, whilst the anæsthetist is outside, but in telephonic communication. The patient lies on the operating table with the head outside and the rest of the body inside the chamber, a rubber diaphragm fitting closely around the neck to exclude entry of air from the outside. A rubber bag communicating with the normal air encloses the abdomen and legs of the patient to prevent the negative pressure exercising its influence on the veins of the systemic circulation.

The great advantage of this method is that not only the lung but the large veins of the chest also are exposed to a negative pressure and are therefore under very nearly normal physiological conditions. The great disadvantages are the expensiveness of the chamber, the space that it occupies, and the fact that it is not portable.

The principle of the hyper-atmospheric methods differs from that of the hypo-atmospheric chamber in that the lung is kept expanded by pressure from within instead of traction from without. Theoretically one might suppose that the results obtained by the hyper- as opposed to the hypo-atmospheric method would be less satisfactory, and that the circulatory disturbances would be considerably greater. It has been found, however, that although the hypo-atmospheric method does undoubtedly produce less disturbance, the hyper-atmospheric methods are essentially practicable and possess certain preponderating advantages.*

Hyper-atmospheric Apparatus.

The forms of apparatus used for the positive inflation of the lung are innumerable, but they may be divided into two main groups :—(1) The closed type, in which the patient respires within a closed circuit ; (2) the open one represented by the various forms of apparatus for intratracheal insufflation.

In the closed type of apparatus, there is a reservoir of air or of a mixture of air and oxygen under pressure. This reservoir is fed by a motor or from compressed gas cylinders. The air is conveyed to the patient either by a casket in which the head is enclosed, by a special closely-fitting mask, by intubation or tracheotomy tubes. The anæsthetic is mixed with the air, or else is given by intravenous infusion. The expired air escapes by another channel in which the pressure is again regulated.

The objections to this type of apparatus are that with the mask or casket, inhalation of vomited matter may easily take place ; and with the intubation or tracheotomy tube, the channel may become obstructed by the secretions of the bronchi or of the lungs. Efficient models are costly and not readily portable. In practice, however, they have been found to be satisfactory and allow free exposure of the lung without risk from the mediastinal flapping.

The intratracheal insufflation or open method differs from the closed in two main essentials : (1) The stream of air under pressure is directed by a catheter into the trachea immediately above the bifurcation of the bronchi ; (2) the excess of air escapes through the glottis around the catheter.

This method was introduced by Meltzer and Auer. The original form of apparatus has undergone constant modification and improvement, but without alteration of the underlying principle. A recent model is shown in Fig. 10, where it will be seen that the apparatus consists of a motor† which actuates a blower and so produces a constant stream of air. This

* These advantages do not apply in those countries where large and completely equipped hospitals exist for rich and poor alike.

† The motor can be replaced by a foot-pump.

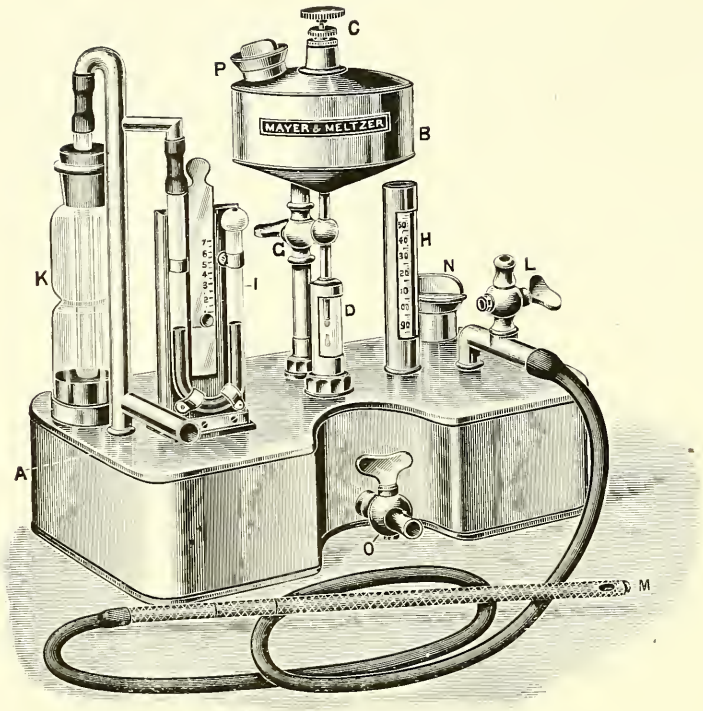

Fig. 10. Dr. Shipway's Apparatus for the Intratracheal Insufflation of Ether.

- A. Air inlet for connection with foot-pump or rotary motor-pump.
- B. Ether container with funnel for pouring in ether and tap for emptying.
- C. Screw tap for regulating flow of ether.
- D. Dropping chamber which allows the flow of ether to be kept under observation.
- G. Equalizing tube with tap for preserving equality of pressure on upper and lower surfaces of ether, thus ensuring regular delivery.
- H. Thermometer.
- I. Mercury manometer with adjustable scale.
- K. Mercury safety valve, should blow off between 20 mm. and 25 mm. of mercury.
- L. Tap for reducing the volume of air stream at intervals.
- M. Intratracheal catheter and tubing.
- N. Filling funnel for hot-water chamber.
- O. Emptying tap for hot-water chamber.
- P. Filling funnel for ether container.

The idea underlying the apparatus is that ether drips from a container into a central chamber, through which passes the air current from the motor or foot bellows, and is there vaporized, partly by this means and partly by the heat imparted by an encircling hot-water jacket. The tap C regulates the flow of ether, which can be delivered in drops or in a continuous stream. The tube G, connecting the ether container and the vapourizing chamber, equalises the pressure on the upper and lower surfaces of the ether, and regular dropping is ensured. During anæsthesia the tap of the equalizing tube must be kept open, but if the container needs refilling during an administration this tap must be first closed before the bung of filling funnel is removed, and kept closed until this has been replaced. At the end of anæsthesia the container can be emptied by means of a tap. The thermometer should register 110 degrees to 120 degrees F., according to the temperature of the room and the nature of the operation. A Y-piece provided with a tap is fixed in the tubing from the motor for attachment to an oxygen cylinder. The hot-water jacket stands in a non-conducting cover. The apparatus weighs $5\frac{1}{2}$ lb. and measures $8\frac{1}{2} \times 4\frac{1}{2} \times 12$ inches.

passes through an ether chamber, a warming chamber, and thence to the catheter, a manometer and safety valve being interposed between the latter two. The pressure, the amount of ether and the temperature can all be regulated. The catheter has two lateral openings at its distal extremity. The average distance in an adult from the incisor teeth to the bifurcation of the trachea is 26 cm., and this length should be marked on the catheter. The diameter should be approximately half that of the glottis. The average size for adults is 22 F. and for adolescents 18 F. The catheter is introduced by direct vision with the aid of a laryngoscope, after the patient has been anæsthetised. The temporary spasm of the glottis which follows the introduction can be abolished by the use of cocaine. It is advisable during the course of the insufflation periodically to cut off the pressure and allow the lungs to collapse, driving out the excess of carbon dioxide.

This method of intratracheal insufflation for the compensation of open pneumothorax entirely supersedes the original forms of closed hyper-atmospheric apparatus. While it has certain advantages over the negative pressure chamber in respect of portability, cost and space, it cannot reproduce the normal physiological conditions with anything like the same approach to accuracy.

With intratracheal insufflation, the breathing is shallow and regular and the movements of the lung therefore do not interfere with the intrathoracic manipulations. The oxygenation of the blood is satisfactorily maintained and at the end of the operation normal respirations recommence almost at once. The pressure can be varied and regulated to any degree required. The catheter rarely produces irritation either of the glottis or trachea. The stream of air, as it escapes, drives out mucus and prevents inhalation of foreign matter, reducing thereby the dangers of the usual post-anæsthetic pulmonary complications. As the electric motor can be easily replaced by a foot-pump, there need be no anxiety in regard to current or fuses. It is possible to keep the patient alive even when both pleural cavities are opened.

In discussing, previously, operations with an open pneumothorax, these were divided into three groups, and it was shown that in group (1) compensation of the pneumothorax was not required; that in group (2) it was not essential, but that in group (3) the operations were of extreme danger if the collapse of the lung and mediastinal flapping were uncontrolled. The comparative simplicity and portability of the intratracheal insufflation method as a means of anæsthetising the patient, and compensating for the pneumothorax, makes it available, and even almost advisable, for all operations in either groups (2) or (3). In general it may be said that, whichever form of apparatus is used, the extent to which the lung is kept expanded can be varied according to the needs of the case, but that the expansion should never be greater than is actually required, since it is not the distension of the lung that is necessary,

but the prevention of the mediastinal flapping. In many cases it is preferable, from the operator's point of view, that the lung should occupy as small an area of the pleural cavity as possible. At the end of the operation, before the opening in the chest wall is finally closed, it is advisable that the pressure be increased so that the lung fills the greater part of the pleural cavity.

Experimentally it has been shown that after an operation with an open pneumothorax, a pleural effusion follows the use of the hyper-atmospheric method of control more frequently than when the lung has been exposed in a negative pressure chamber. This is the commonest and, in fact, almost the only complication of the use of a compensating apparatus in skilled hands.

So far the several forms of differential pressure apparatus have been discussed in connection with the prevention of mediastinal flapping. While this was the object for which they were invented, it does not represent the whole field of their utility. During operations on bronchiectasis, on gangrene and abscess, on phthisis with large cavities full of secretion, or even on hydatids, the contents of the cavities occasionally either burst or are expelled suddenly into the bronchi and so into the trachea. If the patient is unable immediately to expel the discharge by coughing, he may aspirate it into the bronchi of both lungs and produce an instantaneous and total obstruction of them. Death invariably results within a few minutes, since aeration of the lung by natural or artificial means is impossible. Under certain circumstances this disaster is unavoidable. Inversion of the patient during the operation as a prophylactic measure will not avail if the discharge is sufficiently sudden and copious. It is possible that this obstruction of the bronchi of the sound lung can be prevented by intratracheal insufflation. With this method, there is a rapid outgoing current of air along the trachea, whilst in the bronchi the air is under pressure and almost motionless, the exchange of air taking place at the bifurcation of the trachea. It is probable, therefore, that if there is any sudden gush of pus and mucus into the trachea the outgoing current of air will be strong enough to carry this along with it and expel it from the mouth.

Further, the air and secretions in the peripheral parts of the lung are at a slight negative pressure. When an incision is made into an abscess cavity, the air at atmospheric pressure entering through the opening will carry with it the pus into the bronchi. This cannot occur with intratracheal insufflation, as the air within the bronchi is at positive pressure and will therefore rather drive the pus out through the external wound.*

* The tendency will be for the pus to be sucked out when the cavity is opened in a negative pressure chamber.

Chapter III.

DIAGNOSIS AND PROGNOSIS.

The diagnosis of intrathoracic disease is dependent not on the physical signs but on the interpretation of them. Very few, if any, of the signs are peculiar to any one disease, many are common to most. It is only by assembling all the symptoms and physical signs and by a knowledge of the morbid anatomy and mechanical changes that a correct diagnosis is possible.

The morbid changes of the lungs may be considered as they affect the bronchi, the alveoli and the interstitial tissue. Such grouping is no more than a convention for descriptive purposes, as in no chronic or acute disease, except some of the milder forms of the latter (*e.g.*, acute bronchitis), are the changes confined to one single group.

Acute inflammation of the bronchi and bronchioles causes swelling, proliferation and shedding of the epithelial lining, a stimulation of the glands and a general inflammation of the walls. In the very severe cases, ulceration and sloughing may occur; such a condition leads inevitably to changes of the surrounding lung parenchyma. Chronic diseases produce a proliferation and desquamation of the lining epithelium and a general hypertrophy of the whole wall. This hypertrophy is always due to a submucous proliferation and to fibrosis of all the coats: in some cases there is hypertrophy of the muscular wall also. Associated with this hypertrophy of the walls, there is usually an increase in the size of the lumen. Dilatation is not peculiar to the disease bronchiectasis; it is found—not in such a noticeable degree, but sufficiently to produce its own set of physical signs—in all cases of chronic disease of the lung in which fibrosis is the prominent feature

The changes in the alveoli are all of such a nature that they tend to produce diminution or obstruction of air entry into the spaces. This may be brought about by proliferation of the walls and congestion of the vessels, by fibrosis or by the outpouring of exudate, and by the escape of leucocytes and red corpuscles into the lumen.

Fibrosis is the most striking change which takes place in the interstitial tissue. It implicates, either directly or indirectly, all the surrounding lung structures and often spreads to, or is associated with, a similar affection of the pleura. This fibrosis is also responsible for a great part of the mechanical disturbances in the chest.

Each lung is enclosed by walls capable of varying degrees of resistance and on these it exerts constant traction through its elastic fibres, which traction, however, is under normal conditions insufficient to interfere with the movement or position of the walls. The mediastinum is the least, the ribs and cartilages are the most resistant, while the diaphragm is intermediate. When fibrous changes develop in one lung, the state of equilibrium between the lung and the walls is disturbed. The lung is diminished in volume and increased traction is exerted on the surrounding structures, the walls being drawn inwards in proportion to their capacity of yielding. The result is some flattening of the chest wall, raising of the diaphragm, and a, comparatively more extensive, displacement of the mediastinum. The contracting force of the fibrous tissue is directed internally as well as externally, and its most noticeable effects in the former direction are to be found on the bronchi, which become dilated. The converse changes are seen when the pleural cavity, normally a potential space only, becomes filled with fluid or gas. In such cases there is marked displacement of the mediastinum away from that side, considerable depression of the diaphragm and necessarily of the abdominal organs in contact with it, and bulging of the chest wall and intercostal spaces. In addition to this, the lung is collapsed against and displaced with the mediastinum.

The flexibility of the walls, though very striking in some cases, is not sufficient to allow anything like full scope to the efforts at reparation made by the body in chronic diseases. This is shown, on the one hand, by the influence which the fibrous tissue is able to exert on the bronchi, and on the other hand by the inability of cavities to heal by the process of collapse and approximation of their fibrous walls. This fact is, incidentally, an indication of the great difficulty of treating chronic diseases without operative intervention for the readjustment of the pathological mechanical conditions.

Displacement of the mediastinum to the opposite side can be due to pleural conditions only; displacement to the same side to pulmonary conditions only. Increase of lung volume occurs only with emphysema; diminution is found in practically all chronic diseases and also in pneumonia and massive collapse.

A disease may be latent, it may give rise to symptoms without signs, especially when situated deeply, or to signs without definite symptoms; again the manifestation of it may be masked by some concurrent complaint such as bronchitis. The symptoms caused by mechanical changes are proportionate in severity to the rate at which such changes are developed.

The symptoms and signs of pulmonary lesions are due to irritation, infection, obstruction, mechanical changes, cavity formation or to dilatation of existing spaces. The clinical manifestations in pleural lesions are due to irritation, infection and mechanical changes.

Cough is due to irritation of the bronchi or of the pleura; in the former case it is usually associated with an increase in secretion and is an explosive act necessary for the expulsion of such secretion. Coughing is not necessarily an indication of intrathoracic disease. It is a common symptom of morbid changes of the upper air passages, and may be caused by irritation of the lower as well as of the upper surface of the diaphragm. Occasionally there is no obvious source of irritation and the cough is then termed "reflex."

Expectoration. The sputum may be the result of excessive secretion of the bronchial tubes, of exudate into the alveoli or of breaking down of lung tissue. A purulent sputum indicates a secondary infection: an offensive sputum, that there is acute destruction of tissue or secondary infection associated with retention: it is present in cases of putrid bronchitis, bronchiectasis and gangrene.

Blood may come from any part of the respiratory tract. It is present in quantity only in cases where there is destruction of lung tissue or of new growth formation.

Pain is an indication of bronchial or parietal pleural irritation. It is acute in the latter case only. Pleural pain may be local or referred. The reference is to the shoulder in diaphragmatic pleurisy and to the epigastrium when the lower intercostal nerves are involved. In such cases, in association with the referred pain, there may be unilateral rigidity of the muscles of the upper part of the abdomen; this combination may lead to an acute intra-thoracic lesion being suspected at the onset as an acute intra-abdominal one.

"*Tightness*" is a symptom that it is not always possible to differentiate from pain. It is often associated with irritation of the parietal pleura, and may be due to the pain present in such cases or to the pull on adhesions. It is experienced when there is displacement and compression of the lung and mediastinum as in cases of pneumo- and hydrothorax. "Tightness" is commonly complained of in cases of acute bronchitis where there is a considerable obstruction of the bronchi by the viscid secretions.

Dyspnœa. This word is variously used to denote difficulty in breathing of any degree, from that which can be overcome by the full use of all the ordinary muscles of respiration, to that which requires the additional help of all the extraordinary muscles; it is used to denote the rapid respiration such as is seen in pneumonia and to denote also that degree of rapid breathing more commonly referred to as "shortness of breath." The term must be regarded as meaning any or all of these conditions. Dyspnœa is a most important symptom, both from the point of view of diagnosis and prognosis. The conditions which produce it are manifold, but the recognition of the causative factor is often a matter of extreme difficulty. Acute dyspnœa is caused by spasm of the larynx; by sudden obstruction of a large bronchus

(foreign body), or of the bronchioles or alveoli (massive collapse, pneumonia); by acute compression of the lung (spontaneous pneumothorax). The less acute causes are, obstruction of the air entry, either by gradual compression of the bronchus from without, or by obstruction of the bronchial tubes by secretions or neoplasm, or by collapse of the lung, due to hydro-, hæmo- or pneumothorax. Dyspnœa may be due to a diminished capacity for alteration in the volume of the lung during inspiration and expiration, producing thereby a condition of increased air stasis as in emphysema. It is frequently associated with cardiac lesions, especially myocarditis due to toxæmia, and with acidosis in cardio-renal disease. It is present also, though in a less marked degree (and described usually as "shortness of breath"), in cases of fibrosis of the lung in which there is a disproportion in the capacity of expansion of the lung to that of expansion of the chest wall.

PHYSICAL SIGNS.

It is the intention in this chapter not to discuss the physical signs in particular, but rather to draw attention to some of the more important points in the examination of the patient. Many facts can be ascertained by observation only: the general state of nutrition, the presence of pallor, hectic flush or cyanosis, the character of the respirations and of the cough, the odour of the breath, the degree of movement and the flattening or bulging of the chest wall, and the presence or absence of clubbing of the fingers.

The resistance met on percussion is as important as the quality of the note obtained. For the former, the direct method of percussion (finger directly on chest) is the more useful; for the latter, the indirect. All conditions such as fluid, thickened pleura, fibrosis and consolidation which give diminution of resonance, give also increased resistance. The quality of the resistance in cases such as pneumothorax and compression of the lung into the upper part of the pleural space, is often of greater diagnostic value than the quality of the note: in pneumothorax there is the sensation of rebound as from an inflated rubber tube, while when the lung is compressed the resistance is increased above the normal.

The breath sounds may be normal in character but altered in intensity, or they may be abnormal in pitch and rhythm. In the former case the variation is due to changes affecting the conduction of sound, such as fluid or some form of consolidation, or to changes affecting free air entry such as in emphysema. In the latter case, the change is an indication of alteration of the relative size of the air-containing parts as in cavity formation or pneumonia. Rhonchi, râles and crepitations are all indications of an increased exudation. The rhonchi are an indication of viscid mucus in the larger tubes: râles and crepitations, of exudate in the bronchi and bronchioles and in the finest tubes and alveoli respectively.

Vocal resonance affords the same evidence as vocal fremitus and in a clearer form. The alterations in intensity or in the quality of the transmitted voice are brought about in much the same way as are the alterations of the breath sounds. The nasal quality of the transmitted sound, known as œgophony, sometimes heard at the upper border of a pleural effusion, is one of the very few pathognomic signs in intrathoracic disease.

A disease produces by no means homogenous signs over the area affected. Frequently, marked variations in resistance, in the quality of the breath sounds, etc., are found within short distances of each other, as in fibroid phthisis associated with cavity formation. But over the whole of the area affected directly by the disease or secondarily by mechanical changes there will be some alteration of one or more of the physical signs. There are only three pathological conditions in which, as a general rule, there are scattered areas of affected lung tissue separated by areas of normal lung tissue. These diseases are broncho-pneumonia, multiple infarcts and syphilis.

It is a great mistake, during the examination of the chest, to segregate each separate physical sign and, as it were, scatter over the chest a series of labels denoting dullness, bronchial breathing, râle and crepitation. At the end of the examination, the surgeon should be able to assemble all his signs and from the grouping, visualise the morbid processes present in the chest. It may be that the grouping of physical signs suggests the possibility of two different types of change, and in such cases it is necessary to utilise all other possible measures, such as examination of the sputum, radiography and Wassermann reaction. These accessory examinations, with, of course, the history of the case, will in many instances be necessary to determine what is the nature of the disease which is producing the morbid changes, since various infections are capable of producing morbid changes which are closely analogous to each other, and it is these changes rather than the actual disease which are responsible for the physical signs.

The symptoms and physical signs in chronic diseases such as tuberculosis are rarely those caused by the single infection. In the majority of cases, they are due to the changes produced by the tubercle bacilli and to those produced by secondary infection. It sometimes happens that the manifestations caused by the secondary infection completely overshadow those caused by the primary one.

Radioscopy and radiography are of such great assistance in the determination of the character and extent of the lesion that no case should be operated on until a radiogram has been secured, unless, of course, the means for this are not available. It is doubtful in fact as to whether a definite diagnosis or prognosis should be made without the evidence afforded by X-rays. The physical signs reveal roughly the extent of an active lesion, especially of that part which is comparatively superficial: they are also the most certain means at our disposal for recognising the activity or the quiescence of the focus. They do not, however, always afford the necessary

information to enable one to recognise the character of the disease, nor, in certain chronic conditions in which repair by fibrosis has been effected, do they allow of a correct estimate of the damage which has been caused to the lung tissues. A good radiogram will often give just that information which it is so difficult to obtain from the physical signs alone.

Two striking examples may be given as illustrating the value of radiography. The skiagram shown in Fig. 79 (carcinoma of the lung) was taken of a man sent to me suffering from chronic bronchitis and emphysema. The X-ray was taken as part of the routine examination and revealed a tumour of the lower lobe. No symptom or physical sign relative to this tumour could be detected until three weeks later. A woman, aged 35, had whooping cough when seven years old, and ever since she had been under treatment for "indigestion and chest trouble." She was sent to me with the diagnosis of basal pneumothorax and was then X-rayed for the first time. It was at once obvious that all her symptoms and signs were due to a large left-sided diaphragmatic hernia. (See Fig. 36.)

Every patient should be examined with the screen, and a plate taken as well. The screen examination affords evidence of the movements of the diaphragm and of the "lighting up" of the various parts of the lung. The degree of "lighting up" of abnormal shadows with deep inspiration is of particular importance. The screen also gives a general idea of the position and character of the lesion, but does not give details nor permit of any lengthened study. It is for these reasons in particular that the radiogram is required, but in order to be of any use it must be taken with the patient either standing or sitting, and with the breath held. The tube must be soft and the exposure correct. An indifferent radiogram, specially one taken with a hard tube or over-exposed, may be completely misleading.

Prognosis.

In regard to acute diseases, prognosis cannot go much beyond broad conclusions from the condition of the patient and the normal course of the disease. With regard to prognosis in chronic cases, it is possible to make certain general statements. In diseases of the nature of bronchiectasis and in all those in which fibrosis plays an important part, and in chronic pleural effusions, it is impossible to produce more than some temporary improvement by any method of treatment which excludes measures for the readjustment of the mechanical changes. Chronic infective diseases which run their course with little or no rise of temperature are much less amenable to treatment than are those in which there is pyrexia. The prognosis is bad in all cases in which there is cardiac failure, cyanosis, or dyspnœa due to insufficient aeration or diminished alkalinity.

Chapter IV.

DISEASES OF THE PLEURA.

In the two previous chapters, considerable emphasis has been laid on the importance of mechanical changes in association with intrathoracic lesions. These mechanical changes are the most striking manifestations of pleural disease ; they account for the greater part, sometimes for even all, of the symptoms and physical signs, and they are commonly the factor which determines the treatment The importance of them is such that the most satisfactory grouping of diseases of the pleura is one which is based on these changes, as follows :—

Group (1) Local manifestations predominating—acute dry pleurisy.

Group (2) Mechanical changes predominating—pleurisies with effusion, whether simple, tubercular or infective ; pneumothorax including hydro- and pyo-pneumothorax.

Group (3) Local and mechanical combined—tumours.

Acute Dry Pleurisy.

This disease, whatever the actual cause of it, is characterised by one predominating symptom, viz., pain. The pain is due, not so much to the actual inflammation of the pleura, as to the constant irritation of the inflamed area by the movements of the lung. The pain is acute ; it is felt locally or may be referred to the shoulder in cases of diaphragmatic pleurisy, or along the intercostal nerves to the epigastrium. As a result of this reference of pain to the upper part of the abdomen, the condition may simulate an acute abdominal lesion. When not very severe, the pain will yield to complete rest and the application of linseed poultices : in a few cases in which the pleurisy involves the lower part of the chest, relief is obtained from fixation of the chest by strapping. Not infrequently, morphia in repeated doses is the only non-operative measure which will produce relief. The pain, however, can be instantaneously and completely abolished by the injection of oxygen into the pleural cavity. The oxygen is introduced by the same technique as that used for nitrogen and described in Chapter VIII. The gas, by separating the two layers from each other, does away with the friction and therefore with the main cause of the pain. Since the gas is rapidly absorbed, 200 cc. more than is required to abolish the pain are introduced.

A recurrence of pain and the necessity for a second injection are rare. Oxygen exerts a directly beneficial effect on the lesion and for this reason is used in preference to nitrogen.

Cough is frequently associated with the pain. It is usually dry in character, and, when due to irritation of the diaphragmatic pleura, may be continual and distressing. In some cases, the dry pleurisy is the precursor of an effusion. As soon as the fluid has accumulated sufficiently to separate the inflamed pleural membranes, the pain and cough will subside.

Pleural Effusions.

All lesions of the pleura which are associated with the presence of fluid in the cavity have certain characteristic changes in common, and others which are peculiar to the disease. Since treatment, if it is to be efficient, must be directed to the arrest of the pathological processes, to the prevention of further morbid changes and to the removal of or compensation for the abnormal mechanical conditions, it is essential to obtain a clear conception—

(1) Of the effect on the lung and on the surrounding walls of a collection of fluid in the pleural cavity;

(2) Of the importance of the changes which are produced by the fibrinous and cellular contents, when present, of the fluid;

(3) Of the character of the morbid processes which develop in the membranes, particularly in diseases such as tuberculosis.

(1) *The effect on the lung and on the surrounding walls of a collection of fluid in the pleural cavity.*

An effusion, whatever its nature, accumulating in a pleural cavity free of adhesions causes collapse and displacement of the lung in a manner which is constant. The fluid collects at the base of the pleural cavity and displaces the lung at first directly upwards. The upper level of the fluid is almost horizontal,* but is slightly higher at the outer or axillary border.† When the fluid has reached the lower edge of the hilum, the

* It is completely horizontal only when there is gas above the fluid.

† Despite the elastic traction in the lung, the visceral and parietal surfaces of the pleura are not readily separated. As fluid accumulates it falls by gravity to the base of the cavity. With increase in the size of the effusion, the pleural surfaces must of necessity be separated, and the lung, being lighter than the fluid, is displaced upwards; but being fixed at the hilum it must necessarily also be displaced inwards The inward displacement begins at that point on the surface furthest away from the fixed point on which the fluid exerts its pressure, *i.e.*, along the axillary border.

When a healthy lung is displaced by gas, the gas exerts equal pressure on all surfaces, and the lung is eventually collapsed around the hilum, giving the appearance of a pedunculated spherical swelling. This method of collapse, as the result of displacement by gas, is not observed when the lung is the seat of chronic disease: the rigidity of the bronchial tubes prevents uniformity of the collapse and the lung usually becomes flattened against the mediastinum.

displacement of the lung is upwards, but also, to some extent, inwards. With increase of the effusion, this upward and inward displacement continues until the lung lies collapsed against the upper half of the mediastinum and into the extreme apex of the chest. The diminution in the intrapleural negative pressure and eventually the change to a positive pressure, combined with the persistence of the negative pressure in the opposite side, results in a displacement of the mediastinum and its contents away from the fluid. The extent of the displacement is determined partly by the mobility of the structures and varies greatly in different people. When the fluid has accumulated sufficiently to produce a positive pressure, the chest wall becomes gradually expanded into the position assumed during inspiration ; in extreme cases there may even be flattening and rarely bulging of the intercostal spaces. The diaphragm also is displaced downwards : this change is most marked in left side effusions and there is necessarily a similar displacement of the adjacent abdominal organs.

The *physical signs* are therefore :—

(1) Those due to the fluid. There is increased resistance and dullness, most marked at the base and extending higher in the axilla than in front and behind. Shifting dullness, *i.e.*, variation of the level of dullness with alterations in posture, can be obtained with moderate-sized effusions, but only when there are no adhesions between the pleural surfaces. In order to obtain this sign properly, the alteration in posture must be considerable and at least ten minutes allowed to elapse between the two examinations. Owing to the non-conducting quality of liquid, the breath sounds are diminished or absent and the vocal resonance also. At the upper level of the fluid, the vocal resonance may have that nasal quality known as œgophony.

(2) Those due to compression and displacement of the lung. Over the compressed lung, *i.e.*, over the upper and inner part of the chest, resistance is somewhat increased, but the percussion note has a tympanitic quality, known as skodaic resonance. The breath sounds are conducted much more directly from the bronchi to the chest wall, and are therefore bronchial in character and the vocal resonance is increased (bronchophony).

(3) Those due to displacement of the mediastinum. This is recognised by the altered position of the heart's apex beat and of the cardiac dullness.

(4) In large pleural effusions the movements of the affected side of the chest are greatly diminished : there is apparent enlargement of this half of the thorax due to the ribs being forced into the inspiratory position, and there is also obliteration but seldom bulging of the intercostal spaces.

A subcutaneous collection of fluid is rarely seen in association with a pleural effusion except as the result of injury to the pleura by trauma: the commonest cause is puncture of the pleura by a trocar and cannula, especially when two or more punctures are made close together. In empyemata, the pus may escape into the subcutaneous tissues as a result of ulceration of the pleura (see p. 74).

As the fluid is absorbed, or after a simple aspiration, friction sounds may be audible over that part of the chest where the lung has again come into contact with the parietal pleura.

SYMPTOMS.

In quite a number of cases the pleural effusion, developing slowly, produces no symptoms (latent effusion). This is particularly so when the fluid is a transudation of serum secondary to cardiac or renal disease (simple hydrothorax), but may also occur when the effusion is inflammatory or tubercular, whether primary, or secondary to a pneumonia or to phthisis. In other cases, an effusion, developing as a complication of some pulmonary disease such as pneumonia, causes an accentuation only of symptoms already present. Such a change may then be erroneously attributed to an exacerbation of the primary lesion, and unless the chest is carefully examined, the presence of the fluid is not recognised.

Dyspnœa, a feeling of tightness in the chest, and fever are the three chief symptoms of pleural effusion. The intensity of the dyspnœa is directly proportional to the rate of the accumulation rather than to the amount of the fluid.*

Fever is present in most cases which are of an inflammatory origin, the temperature rising usually to 100° or even to 102° F. and showing a diurnal variation of 1 to 2 degrees. Such a temperature is no guide to the character of the effusion. A high swinging temperature suggests a pyothorax, but a pyothorax may be afebrile. The rate of the pulse is increased, owing either to the febrile condition or to the displacement of the heart. When the effusion is large, the patient is unable to lie on the sound side.

The amount of fluid present in the pleural cavity determines the physical signs, whilst the symptoms are mainly dependent on the rate of accumulation of that fluid.

* In a patient confined to bed, this symptom may be inappreciable even when there is a very large purulent effusion.

(2) *The importance of the changes which are produced by the fibrinous and cellular contents, when present, of the fluid.*

Effusions develop most frequently in association with pneumonia or pulmonary tuberculosis, but they may complicate any disease of the lung, of the mediastinum, or of the pleura itself. They may be caused by the invasion of the pleura (independently of the lung) by pyogenic or non-pyogenic organisms such as the streptococcus, staphylococcus, bacillus coli, pneumococcus, tubercle bacillus or gonococcus. In these cases the condition is usually a complication or sequela of some acute specific (including influenza), of abdominal suppuration, particularly appendix abscess, or of local trauma. Occasionally there is no recognisable preceding infection. Effusions may occur as a result of irritation produced by inflammation of adjacent parts, *e.g.*, pericarditis and subdiaphragmatic abscess, or of direct extension from these. The effusion may be the transudation of non-inflammatory fluid (hydrothorax) secondary to cardiac failure, renal disease or cirrhosis of the liver.

The character and appearance of the fluid varies greatly in the different cases. It may be practically devoid of any cellular or fibrinous content and is then of a pale straw colour or almost colourless as in a hydrothorax; on the other hand, it may be opalescent and contain much solid matter in the form of shed epithelial cells, lymphocytes and fibrin, as in some tubercular effusions, or be almost solid with fibrin and lymph, as in some pneumococcic infections. All intermediate degrees between these extremes are to be found. The lymph and fibrin, when present, together with the cells which are entangled in them, are deposited on the visceral and parietal pleura and fill up and gradually obliterate the sinuses. If this state of affairs is allowed to persist and the primary inflammation continues, the deposit is gradually organised and the pleural membranes are progressively thickened. This fibrosis, together with the obliteration of the sinuses and the necessary consequent fixation of the lung, mediastinum and diaphragm, will then become permanent.

The character of the deposit and the duration of the acute inflammatory processes are of much greater importance in determining the extent and the permanence of these changes than is the length of time during which the lung is collapsed or the pleural cavity is occupied by an effusion. A healthy lung may be kept collapsed for months or even years by a simple effusion, and yet be quite capable of complete re-expansion: on the other hand, if the inflammation of the pleural membranes continues active and the organisation of the deposited lymph is rapid, re-expansion of the lung at the end of even a month may be quite impossible.

The character of the effusion is therefore one of the main determining features in the treatment of the case.

(3) *The character of the morbid processes which develop in the membranes, particularly in diseases such as tuberculosis.*

Infection of the pleura by tubercle bacilli causes certain special morbid changes which are of fundamental importance. These changes are quite different in character from the inflammatory processes, but are peculiar to those infections which are characterised by the formation of granulomata (tuberculosis, syphilis, actinomycosis). The granulomata are small tumours consisting of granulation tissue which have been produced by the healthy tissues as a reaction to the organisms. The function of this granulation tissue is to destroy the organisms and then to become converted into fibrous tissue. In favourable circumstances, this change actually does take place and the disease is arrested. If the conditions necessary for the success of the granuloma are not present, it will steadily increase in size, attacking and destroying the surrounding healthy tissues. With increase in size there is progressive decrease in the efficiency of the blood supply to its centre. The natural consequence of this is that the tumour caseates, liquifies and finally breaking through on the inner surface of the pleura becomes converted into an ulcer. The granuloma of tubercle differs from others in that its progress through dense fibrous tissue such as the pleura is strongly resisted.

So long as the tubercular infection of the pleura has not progressed beyond the granuloma stage, the effusion closely resembles in character that of a simple pleurisy, and is usually sterile. But when the granulomata have undergone the changes of caseation and ulceration, the fluid is full of pus cells and of debris, while the walls are very greatly thickened and fibrosed ; the surface moreover is sometimes so vascular that a simple puncture may give rise to considerable bleeding. In such cases, the fixation of the lung and of the chest wall is absolute.

These changes have a special bearing both on the treatment and on the prognosis.

TREATMENT.

It follows from what has been said above that a hydrothorax which has been absorbed or removed leaves behind it no permanent changes either of the pleura or of the lung, as there has been no irritation of the walls of the pleural cavity, no deposit on the membranes and no debris which can obliterate the pleural sinuses. The duration, moreover, of the hydrothorax is of no ultimate consequence provided the lung is not diseased, since the healthy lung is at any time capable of complete re-expansion.

An inflammatory or tubercular effusion, on the contrary, invariably leaves some change behind it : there is thickening of the membranes, there is a deposit of debris at the base of the pleural sac and in the pleural sinuses, and consequently there is a partial or even complete interference with the

free re-expansion of the lung.* The thickening of the pleura is most marked when there has been long standing chronic disease of the membranes (tuberculosis), and when the formation and deposit of fibrin has been excessive (pneumococcal infection). In the latter type of case, the permanent changes may develop with considerable rapidity.

It is commonly regarded as a satisfactory conclusion to a pleural effusion if the whole of the fluid disappears and the pleural membranes regain contact with each other. In the majority of those cases in which such a conclusion has been reached after an interval of as much as a month or more, the patient is not cured in the sense that he is as well as he was before the onset of the pleurisy. He may feel well and be equal to his work if his occupation is a sedentary one, but he is easily fatigued and as a general rule it will be found that some degree of shortness of breath is still present on exertion. This symptom may be quite slight, but on the other hand, it may be sufficient in intensity to interfere with his work and pleasures, if these are in any way active. The cause of the persistence of this symptom is the pathological changes noted above. This shortness of breath is in all probability a reflex condition dependent on the existence of an adverse ratio of capacity for expansion of the chest wall and for expansion of the lung.†

Hydrothorax.

The treatment of hydrothorax is that of the disease which is responsible for it. Only when the fluid is sufficient to produce any symptom of discomfort, such as shortness of breath, are active steps indicated. The fluid must then be removed by aspiration with oxygen replacement (*vide infra*).

Inflammatory (non-purulent).

Many simple effusions are absorbed spontaneously; that is to say, that the acute stage subsides within a few days and the processes of absorption are then able to deal adequately with the fluid accumulated, the whole being absorbed within a fortnight or three weeks. In these cases no surgical treatment is required. When, however, the processes of absorption are delayed, operative intervention is indicated.

The line of treatment which should be adopted in all cases is as follows :— As soon as an effusion is diagnosed, the character of the fluid must be ascertained by withdrawing some of it for examination. If the fluid is found

*Partial interference with the free re-expansion of the lung does not necessarily imply that the visceral and parietal pleura are incapable of coming into complete contact.

† This shortness of breath is found in cases in which there is no associated fibrosis of the lung, as shown by X rays; in cases in which there is no emphysema of the lung or deficiency of movement of the chest wall on the same side, or diminution of lung area. A similar shortness of breath develops also during the period of absorption of gas in a case under treatment by nitrogen displacement and can be instantly abolished by the introduction of more gas into the pleural cavity.

to be purulent, the pleural cavity must be at once drained (*vide infra*). An effusion which proves to be tubercular must be aspirated by the oxygen replacement method with as little delay as possible. A simple inflammatory effusion, provided it is of quite recent onset, may be watched. At the end of ten days, if there is no clear evidence of absorption, aspiration by the oxygen replacement is indicated. During this period of waiting, if there is the slightest suggestion of a possible change in the character of the fluid, a further sample must be withdrawn for examination.

Method of Withdrawal of Fluid for Purposes of Examination.

A glass Record syringe of 5 c.c. capacity, a hypodermic needle, a second needle at least 5 cm. in length and with a correspondingly larger bore, and a small supply of 2 per cent. novocaine, are required. A small area over the seat of the effusion, preferably in the mid-axillary line, is painted with iodine. About 1 c.c. of novocaine is drawn up into the syringe, which is fitted with the smaller needle. The point of the needle is driven obliquely into the skin and sufficient novocaine is injected to produce a wheal 1 cm. across. The needle is withdrawn, thrust through the centre of the wheal at right angles to the skin, and pushed slowly inwards, novocaine being injected all the time. Slight pain may be felt as the needle touches the pleura, in which case a little extra novocaine is injected at that point, so as to ensure complete anæsthetisation of the membrane. The needle is withdrawn, removed from the syringe, the larger one substituted and driven in up to the hilt along the anæsthetised track. Fluid should now enter the syringe on withdrawing the plunger. Failure to tap the fluid when such is present means either that the spot chosen is wrong, or that the point of the needle is obstructed by fibrin, or has been driven through a thin layer of fluid into the lung.

ASPIRATION BY THE OXYGEN REPLACEMENT METHOD.*

A small recent effusion of not more than 700 or 800 c.c. of fluid can be removed by simple aspiration. But aspiration without oxygen replacement of a larger effusion is incomplete, generally causes the patient pain or considerable discomfort at and after the aspiration, and is not entirely devoid of danger.

Aspiration with oxygen replacement is devoid of danger or pain: it enables the whole of the fluid to be withdrawn at one sitting: it renders

* The complete removal of pleural effusions with the regulation of intrathoracic pressure by oxygen during aspiration (oxygen replacement) was first introduced by me in 1912 (*The Lancet*, December 28th, 1912). This method did not gain much acceptance until the present war, when the great advantages of it in the treatment of hæmothorax became generally recognised.

The use of air to facilitate the withdrawal of fluid has been periodically advocated since 1882, but the advantages of oxygen and of complete control of intrathoracic pressures do not appear to have been previously recognised.

possible the radiological examination of the whole pleural cavity and lung: by virtue of the oxygen it tends to inhibit the recurrence of the effusion: it permits of the complete regulation of the intrapleural pressures.

The accompanying three skiagrams of the same patient (Figs. 11, 12, 13) illustrate some of the disadvantages of simple aspiration, and the advantages of aspiration with oxygen replacement. Fig. 11 was taken before and Fig. 12 after ordinary aspiration. When 1,200 c.c. of fluid had been removed, the patient complained of pain and the needle had to be withdrawn. It will be seen in Fig. 12 that the level of the fluid is about $1\frac{1}{2}$ intercostal spaces lower than in Fig. 11, and that the mediastinum has returned to a more normal position. There is still, however, a considerable amount of fluid in the pleural cavity, and one object for which the aspiration was done, namely, to obtain a clear X-ray of the lung in order to confirm the diagnosis of malignant growth, has not been attained. Fig. 13 was taken six days later after a further 1,500 c.c. of fluid had been removed by oxygen replacement, without causing the patient any discomfort. In this skiagram not only the tumour but the whole of the diaphragm are visible.

Technique. A Record syringe, a hypodermic needle, 2 per cent. novocaine, a double-edged tenotome, a Potain's aspirator and the apparatus for introducing the oxygen and controlling the intrathoracic pressure, are required.

The apparatus which I have devised for oxygen replacement is shown in Fig. 14. It consists of a box* containing a manometer, filter and container. The manometer is connected by a T-piece, in the one direction (through a filter) to the needle, and in the other, by way of a stop-cock with the side opening near the base of the container. There are two other openings in the container, the lowest being connected with an oxygen cylinder, and the uppermost being open to the air. During oxygen replacement a stream of oxygen flows steadily through the container, filling it and escaping at the upper opening. If the needle is in the pleural cavity and the tap between it and the container is open, oxygen is sucked into the cavity (passing through the filter) when, and as long as, the intrapleural pressure is negative and at a rate proportionate to the pressure. The needle has two taps; the one, as in Potain's cannula, to exclude air after removing the stilette, and the other to prevent the entrance of fluid along the needle and tube when the pleural effusion is at a positive pressure.

* The box measures 27 × 11¼ × 4 inches. The front and right side are both hinged and close on to the base so that when open there is a wide base for support and free access to all the contents.

Round the left side and base are (shown in dotted lines) all the parts of the Potain's aspirator, except the bottle. There are also compartments for the tenotome, for two Record syringes of different sizes, various needles and two glass-stoppered bottles, one for novocaine and one for iodine.

Fig. 11. Case J. O. October 19th. Before treatment. Aspiration was necessary in order to relieve symptoms and for diagnostic purposes.

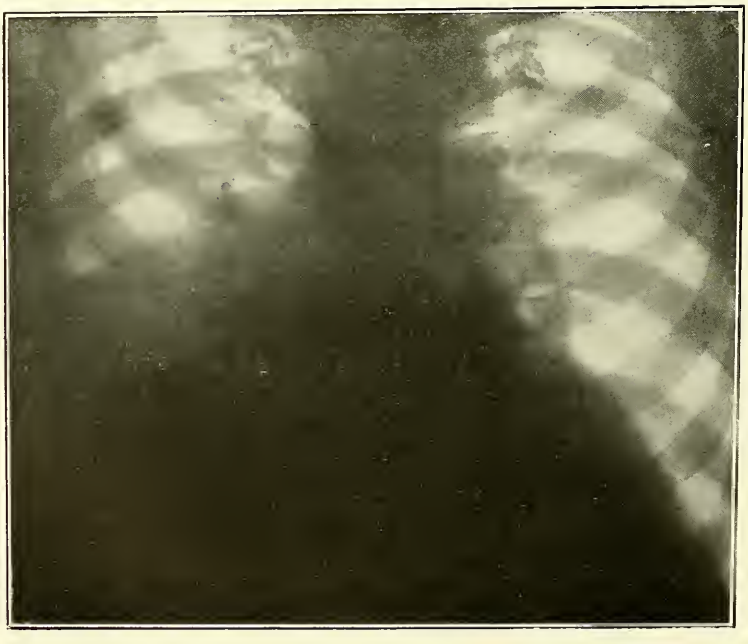

Fig. 12. Same case. October 20th. Skiagram taken within twelve hours after the removal of 1,200 c.c. of fluid by simple aspiration. Further withdrawal, on this occasion, was impossible owing to the development of acute pain and cough.

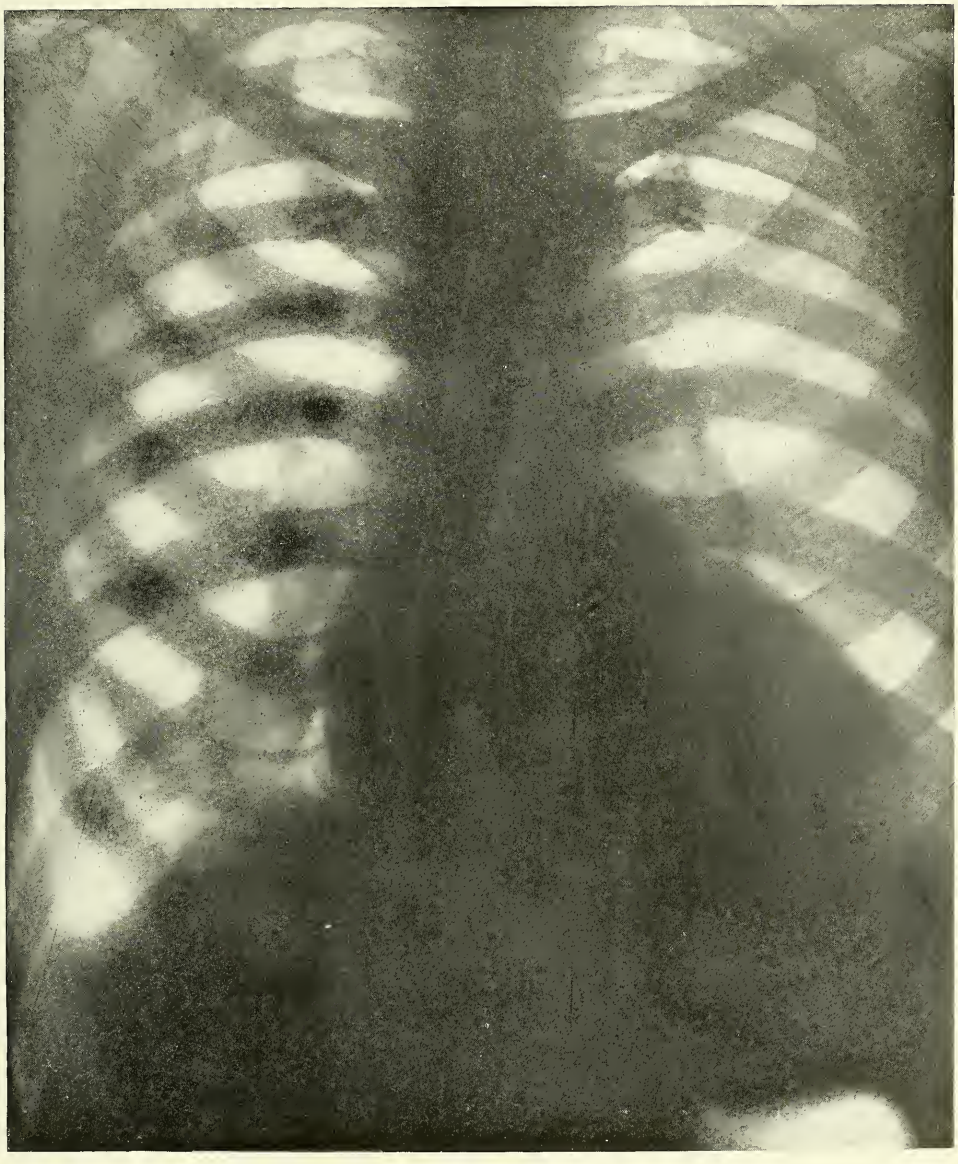

Fig. 13. Same case. October 26th. Skiagram taken twelve hours after the complete removal of the fluid by oxygen replacement. 1,500 c.c. of fluid were withdrawn without causing any symptoms of distress. The whole of the lung is rendered visible, and the tumour, situated in the right lower lobe, can now be seen.

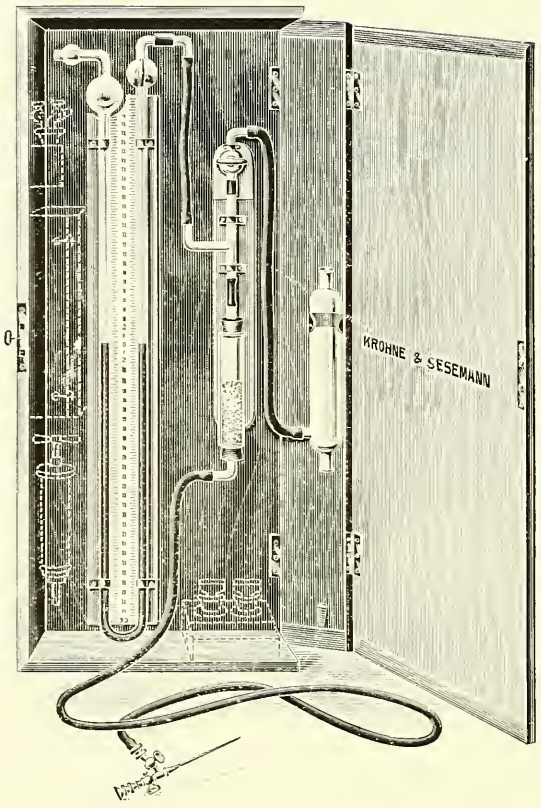

Fig. 14. Author's apparatus for oxygen replacement of pleural effusions.

The patient should be sitting up in bed in a comfortable position and supported by plenty of pillows, none of which should project beyond the back on the affected side. The arms should be raised and supported. The whole of the outer side of the chest (in the case of a large effusion occupying the greater part of the pleural cavity) is painted with iodine. The Potain's aspirator is meanwhile connected up and tested with water to ensure that it is working* and the sterilised filter, rubber tubing and cannula are connected to the oxygen apparatus. The skin and deeper tissues are infiltrated† at

* When buying a Potain's aspirator, two parts need special attention. The pump usually supplied has two nozzles to it, one at the end and one at the side. This is a source of great danger, as if the bottle is connected with the end nozzle (the more obvious one) and the apparatus is not first tested, a positive pressure will be produced in the bottle on pumping, and air driven into the chest when the connecting tap is turned on. The single nozzle vacuum pump is much safer. The openings in the stop-cocks are frequently almost half the size of the largest cannula, so that flakes of fibrin which will pass through the latter will be held up and obstruct the former. These openings should be specially bored out.

† The procedure is the same as that described on p. 46 for exploratory puncture. The anæsthetisation of the track of the cannula is of considerable importance, owing to the dangers of pleural reflex (see p. 89). Death from this cause at the moment of puncture of the sensitive pleura does occasionally, though rarely, occur.

two places, one in the 7th space in the mid-axillary line, and the other two spaces above it.* The skin, in the centre of the infiltrated areas, is punctured with the double-edged tenotome ;† the trochar and cannula of the aspirator is driven slowly but steadily through the lower anæsthetised area inwards and slightly upwards, and the trochar withdrawn. The cannula connected with the oxygen apparatus is then driven directly inwards through the upper anæsthetised area and its trochar withdrawn, both taps on the cannula being turned off. Aspiration is now begun. After about 500 c.c. of fluid have been aspirated, the tap on the oxygen cannula connecting it with the manometer is turned on and a slight negative pressure will probably be registered.‡ The tap between the oxygen container and the cannula, which up to the present has been shut, is now turned on fully, and, until the end of the procedure, is turned off only when aspiration is temporarily stopped in order to produce a fresh vacuum in the bottle. During the aspiration it will now be found that the manometer shows a pressure during expiration varying between zero and -2 mm. of mercury, and during inspiration between -3 and -6 mm. of mercury.‖

Towards the end of the aspiration, oxygen will be drawn off with the fluid. At this stage the Potain's cannula should be depressed into the posterior costo-phrenic sulcus. Whenever the sizzling sound indicating the drawing out of the gas is heard, the aspiration must be momentarily stopped, as the outflow, owing to the larger size of the cannula, is more rapid than the inflow through the oxygen cannula, and the negative pressure rises rapidly.

The final stage is the adjustment of the pressures. The inflow of oxygen is discontinued and the gas is slowly drawn out until the patient complains of a feeling of tightness. The aspiration is then stopped, the tap controlling the oxygen is turned on for a few seconds until the sensation of tightness disappears ; both cannulas are then withdrawn.§

The intrapleural condition at the end of aspiration with oxygen replacement of a recent large effusion is as follows :—There is a negative pressure equal to about 10 mm. of mercury. The lung occupies the greater part

* This region is chosen as the ribs and spaces are easily recognised owing to the absence of thick muscles covering it (see Fig. 4).

It is a useful practice, as soon as the 2nd area has been infiltrated and before pulling out the needle, to see, by withdrawing the plunger, whether fluid is present at that spot. Another syringe and needle can then be used for the anæsthetisation and exploratory puncture in the lower area.

† When the skin is not incised, it offers considerable resistance to the trochar and cannula. The force required to pierce the skin frequently carries the cannula with a jerk on to a sensitive rib, causing pain and shaking the patient's confidence. The drag of the cannula through the skin may carry organisms with it from the surface into the pleural cavity.

‡ If this tap were turned on earlier, fluid would be forced up into the tubing and would interfere with the manometric readings.

‖ The manometer contains water ; the scale is divided up so as to read the equivalents in mm. of mercury, as intrapleural pressures are usually spoken of in those terms.

§ The only dressing required over the puncture holes is a small piece of zinc oxide plaster. This, however, must be removed the next day, otherwise the wounds will suppurate.

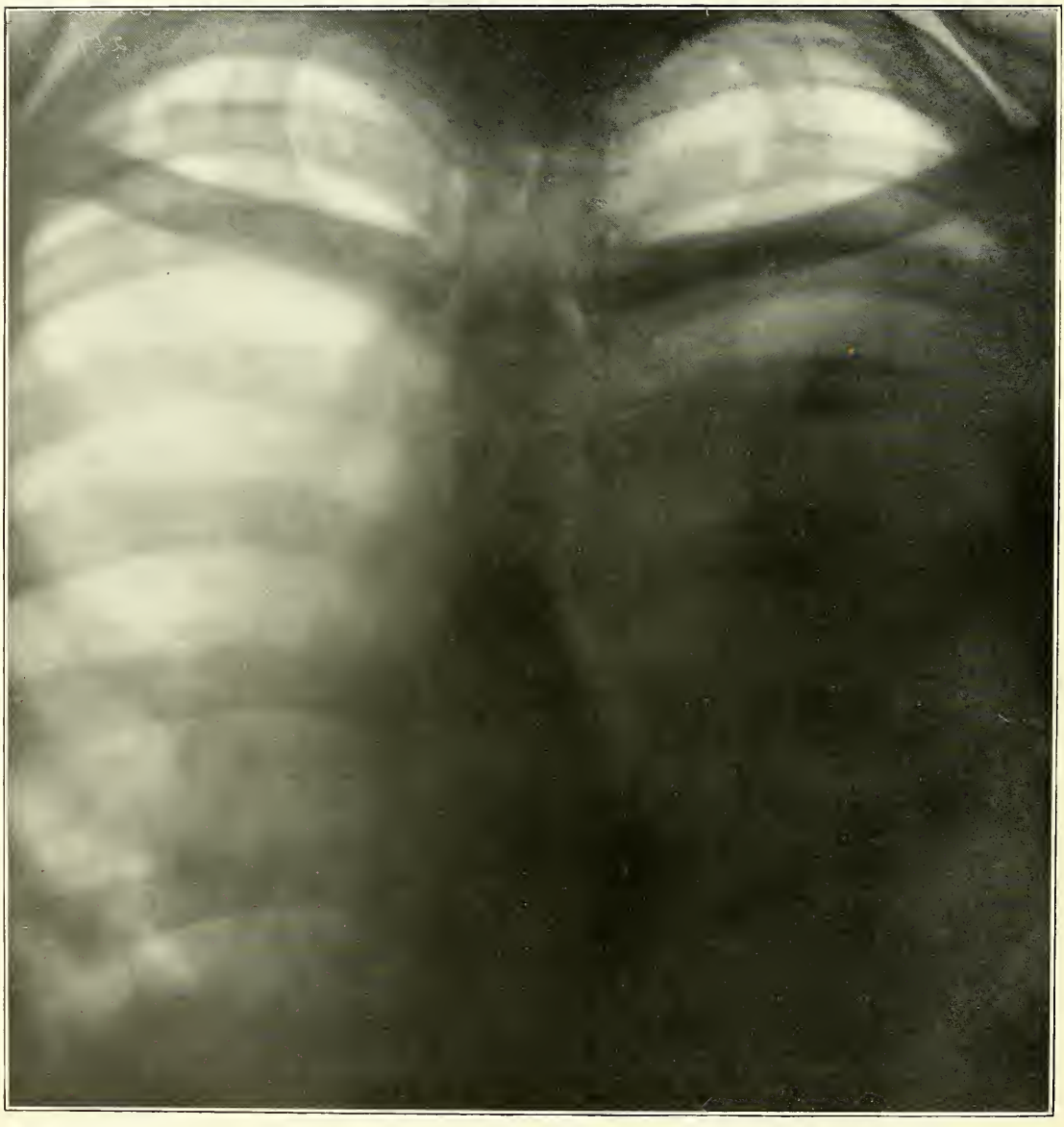

Fig. 15. Case G. N. Tubercular effusion. Left side. November 12th, 1911. This patient had been tapped repeatedly between May and November, but there was always rapid re-accumulation of the fluid.

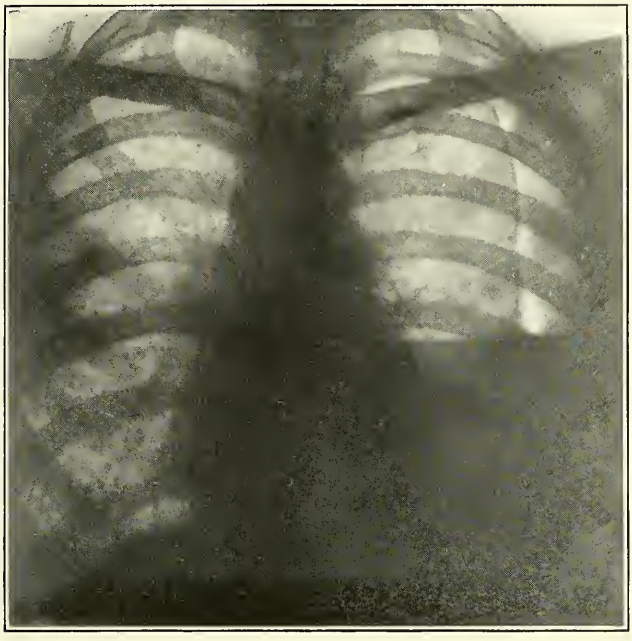

Fig. 16. Same case. Skiagram taken November 17th. The fluid had been removed by oxygen replacement on November 13th. This is the first case in which this method of treatment of effusions was used.

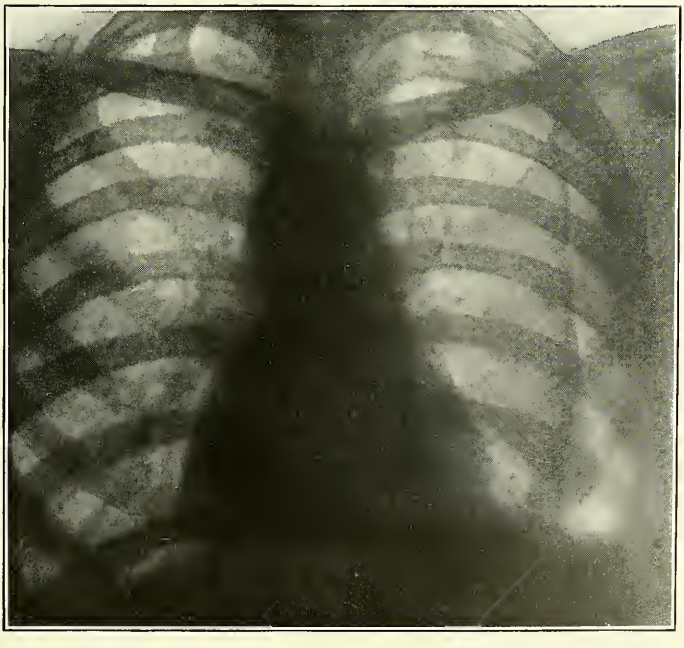

Fig. 17. Same case. December 14th. This shows a later stage in the expansion of the lung by oxygen replacement.

of the pleural space, but does not entirely fill it. There are a few c.c. of fluid left* and some 300 to 500 c.c. of oxygen. During the ensuing few days the oxygen is absorbed and the lung, continuing to expand owing to the pull on it exerted by the negative pressure, occupies in part the space previously taken by the gas ;† the rest of the space is occupied by nitrogen and carbon dioxide gas.‡ These are in turn absorbed, but at a less rapid rate.

In cases of chronic effusion two difficulties have to be contended with—the recurrence of the effusion and the resistance to expansion of the lung owing to the thickening of the pleura and adhesions. There is no doubt at all that the oxygen helps materially, but does not entirely prevent the formation of a fresh effusion. More than one aspiration with oxygen replacement may be necessary. On each occasion the amount of fluid to be withdrawn will be less, and the negative pressure which can be tolerated will be higher.|| This will enable a steadily increasing traction to be exerted on the lung. As a general rule, three or four aspirations are all that prove necessary ; but in one case of mine, a chronic tubercular empyema of seven years standing, eight aspirations with oxygen replacement, extending over a period of eight months, were required to get the visceral and parietal pleura in contact.§

* When oxygen flows into the pleural cavity, the point of the needle being in the fluid, the upper portion of the fluid is frothed by the gas. For this reason, in cases of hydro- or pyo-pneumothorax, the oxygen needle is always introduced into the gas if possible.

† In a quite recent case it may be possible for the lung to expand at the same rate as the oxygen is absorbed. In chronic effusions, the two are never commensurate.

‡ In February and March, 1912, Sir William Ramsey very kindly examined for me several samples of gas taken before and during treatment by oxygen replacement of a case of pyo-pneumothorax :—

February 21st, before treatment— N_2 95·45 %
CO_2 4·55 %
No other gas was present.
February 24th, after withdrawing 1,250 c.c. of fluid and gas and running in 750 c.c. of oxygen—
N_2 74·09 %
O_2 21·56 %
CO_2 4·35 %
March 4th, before treatment on that day—
N_2 98·13 %
O_2 0·69 %
CO_2 1·18 %
After replacing the gas by 1,150 c.c. of oxygen (there was no fluid)—
N_2 76·34 %
O_2 20·71 %
CO_2 2·95 %

|| It is surprising what a very high negative pressure can be tolerated provided that it is produced gradually. In one of my patients I have produced at the seventh aspiration (six months after the first) a negative pressure equal to considerably more than 60 mm. of mercury (which was the highest I could record on my manometer), without his suffering the slightest discomfort. This patient could work with a pressure equal to −50 mm. of mercury. At the first aspiration, however, a patient can rarely tolerate without pain a negative pressure equal to more than 15 mm. of mercury (see Fig. 18).

§ In this case the visceral pleura was too thick to allow the lung on the affected side fully to expand. In order to get the two pleural membranes in contact, it was necessary to cause displacement of the mediastinum to that side. This, however, had no deleterious effects. Dr. Thomas Lewis kindly examined this patient and took electro-cardiograms before and after one of the later oxygen replacements, during which the heart and mediastinum, already drawn over to the affected side, were displaced a further inch. He was not able to find any evidence of interference with the heart's action (see Figs. 19, 20, 21).

A chart showing the varying pressures and the amount of fluid removed and skiagrams of this patient are shown in Figs. 18, 19, 20 and 21.

Whenever there is gas in the pleural cavity together with fluid, whether it has been introduced from outside or is the result of rupture of the lung,

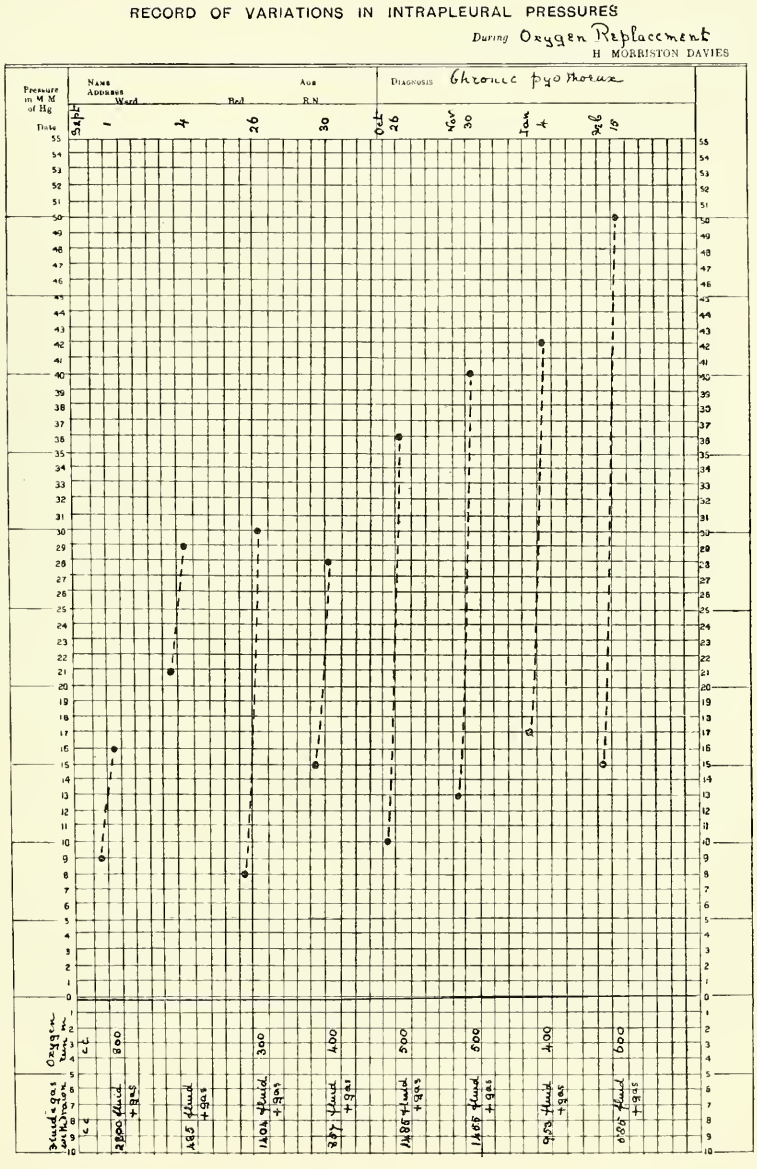

Fig. 18. Case E. B. Chart showing variations in intrapleural pressure at the beginning and at the end of eight aspirations with oxygen replacement

both the gas (consisting of nitrogen and CO_2) and the fluid must be replaced as far as possible by oxygen, so as to get the direct beneficial effect of the oxygen, and, by the rapid absorption of it, to maintain a continual traction on the lung.

When the effusion is associated with active pulmonary tuberculosis, it is advisable that the fluid should be removed, but that the lung should be maintained in a collapsed condition for a considerable period of time (see

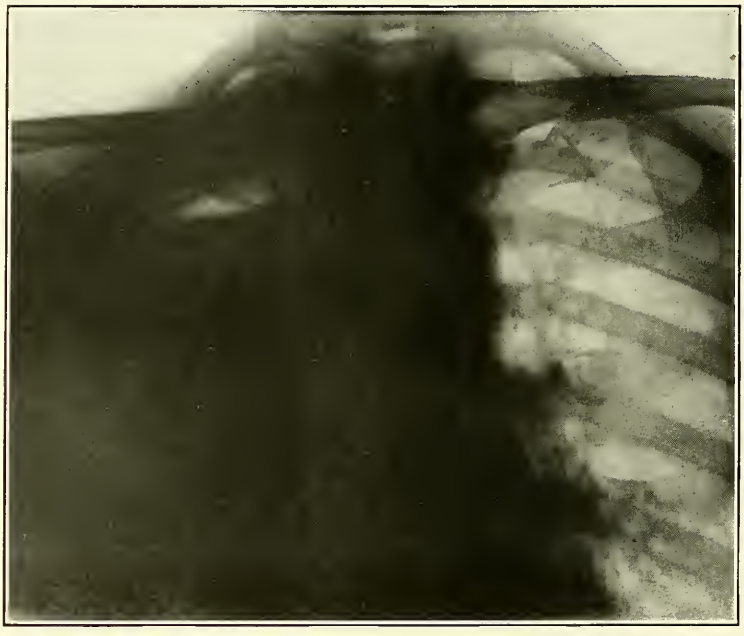

Fig. 19. Case E. B. Chronic pyothorax. Right side. September 1st, 1913. The chest had been tapped three days previously and 2,350 c.c. of fluid had been removed. After the radiogram had been taken, a further 2,800 c.c. of fluid were removed by oxygen replacement.

Chapter VIII). The fluid is at first replaced by oxygen, but a slight positive, instead of a negative, pressure is left in the pleural cavity. As soon as the recurrence of the fluid has been stopped, nitrogen is injected to make up for the loss of volume of the intrapleural gas due to the rapid absorption of the oxygen by the tissues. The nitrogen is not used at the beginning, as it does not possess the therapeutic advantages of oxygen.

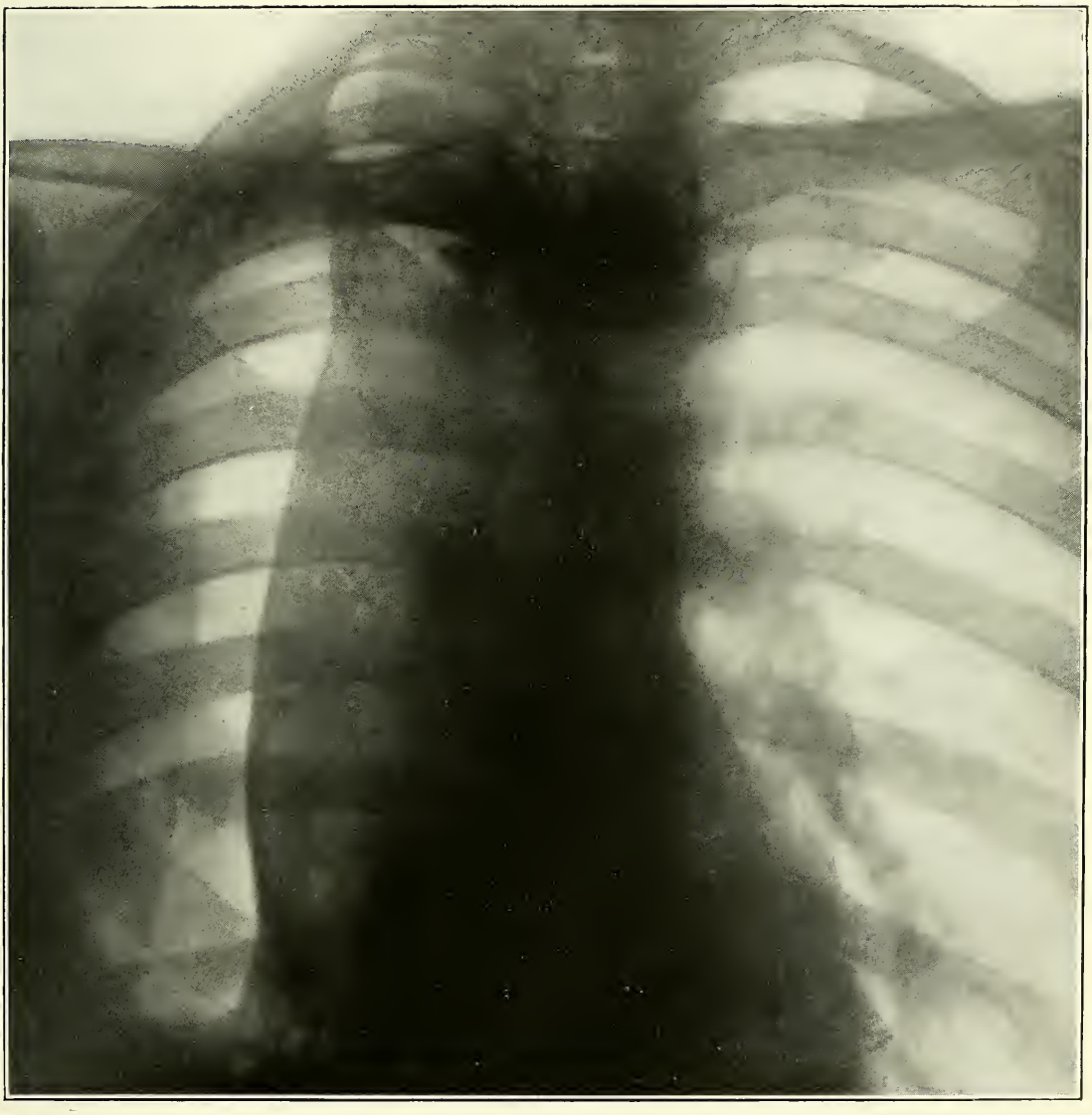

Fig. 20. Same case. October 1st.

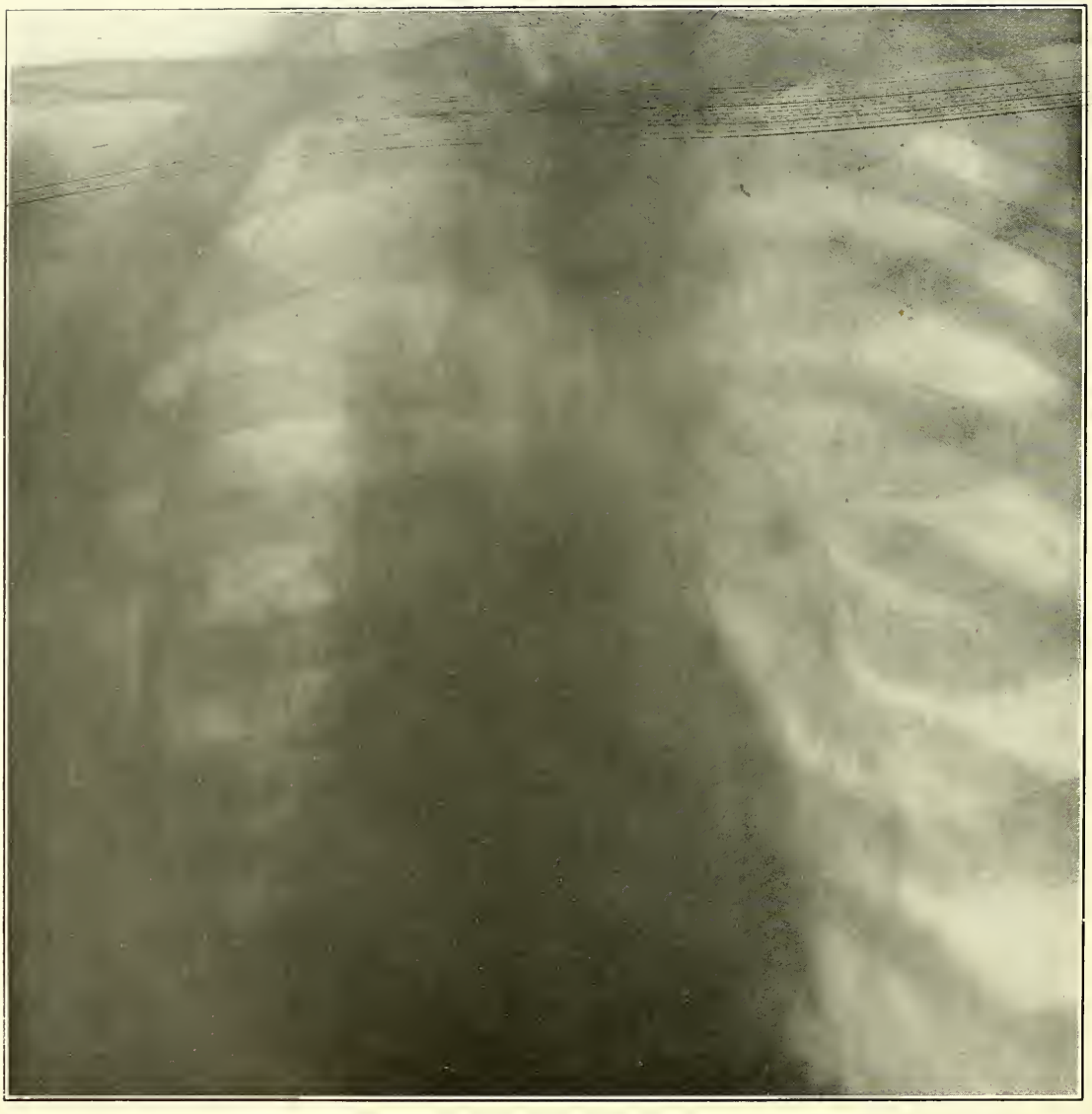

Fig. 21. Same case. Radiogram taken after the sixth oxygen replacement. The general opacity on the right side is due to the thickened pleura, and the obscurity at the base is due to the cellular debris.

TREATMENT OF PLEURAL EFFUSIONS BY PLEUROTOMY.

It has already been stated that in some varieties of pleural effusion there is a considerable deposit of fibrin. This fibrin may so continuously block the cannula during aspiration as to render such method of treatment impracticable. In order to remove this fibrinous effusion, it is necessary to make an opening into the chest, which opening must be closed immediately after the removal of the fluid and deposit. The risk of this method is that the fluid may leak out through the wound and form a sinus; this will lead to secondary infection of the pleural cavity and a chronic discharging empyema. The danger of sinus formation can in most cases be averted by making a high opening into the thorax, by closely stitching up the divided pleura, by maintaining from the time of the closure of the wound onwards a high negative pressure in the pleural cavity, and by keeping the patient during the ensuing ten days after the operation propped up in bed so that the fluid which re-accumulates in the intervals between the aspirations with oxygen replacement never comes into contact with the deep line of sutures.

Technique. Half an hour before the operation the patient is given a third of a grain of morphia and the tissues in the region of the incision (the 4th rib from mid to anterior axillary line) are anæsthetised by regional anæsthesia with 4 per cent. novocaine and adrenalin.* The patient is placed on the table in a semi-recumbent position with the affected side projecting slightly over the edge. An incision is made over the 4th rib, the periosteum is reflected and about 7 cm. of bone are removed. An incision is then made through the internal periosteum and parietal pleura. The division of the periosteum should not reach up to either cut end of the rib, and the whole wound must at once be completely covered by gauze. The gloved finger is inserted through the opening so as to control at first the rate of escape of the fluid,† and at the same time to set free as much of the deposited fibrin as possible. The fibrinous deposit in the more distant parts of the cavity must be gently detached and removed with the aid of moist gauze mops held in sponge forceps.

When the pleural cavity has been emptied, the margins of the wound are carefully cleaned and catgut mattress sutures are passed through the internal periosteum and pleura on either side of the incision. In order to avoid tension on these stitches, the adjacent ribs are approximated by silver wire. The mattress sutures are then tied and the external periosteum and the adjacent intercostal muscles on either side are brought together over them by a second row of stitches; the cutaneous incision is closed.

* If there is not an expert in regional anæsthesia at hand, it is wiser to infiltrate the tissues locally one hour before the operation.

† A precautionary measure to avoid the shock caused by the rapid reduction in intrapleural pressure and the sudden alteration in the position of the mediastinum.

The air in the pleural cavity must now be changed for oxygen* and as high a negative pressure left as the patient can tolerate with comfort.†

Following on this operation, there is always some re-accumulation of fluid. As it is essential that the pressure in the pleural cavity should never be positive and that the fluid should not come in contact with the wound, the patient must be kept propped up in bed and the fluid drawn off by oxygen replacement not later than the third day after the operation (earlier if the effusion is acute and excessive). On each occasion the negative pressure left in the pleural cavity must be as high as can be tolerated.

Tubercular Pleurisies.

Effusions due to infection of the pleural membranes by tubercle vary considerably in character. The fluid may be pale yellow and translucent, may be opalescent, or may be thick with pus cells and debris (tubercular pyothorax). As has already been stated on p. 44, this difference depends on the efficiency of the granuloma as a protective mechanism.

There are three essentials in the treatment of tubercular pleurisies :—

(1) The increase of the resistance of the patient to the disease by hygienic measures.

(2) The removal of the fluid by aspiration with oxygen replacement.

(3) The granulomata must be placed under conditions which are most favourable for the accomplishment of their object, *i.e.*, the destruction of the tubercle bacilli and the subsequent conversion of the granulation into fibrous tissue. In this connection, two important facts must be borne in mind : Firstly, movement is inimical, and rest most beneficial to the success of the granuloma : secondly, the pleura is extremely resistant to the destructive effect of the granulation tissue when the tumour, owing to the virulence of the organism or the lack of resistance of the tissues, has passed from its benign and protective state to a malignant and destructive one.

For the purposes of treatment, three types of pleural effusions must be considered. The grouping of these types is determined entirely by the changes in the pleura.

In the first type, the morbid processes may be described as minimal. The pleural membrane is dotted with granulomata, there is a certain degree of fibrosis but no ulceration. The fibrosis is not sufficient to interfere with the re-expansion of the lung by traction, such as can be produced by oxygen replacement. In this way it is possible to bring the visceral again into

* This is done by the same technique as for oxygen replacement of fluid. The finest Potain's cannula is used.

† Not equal to more than 12 mm. of mercury if the patient is still under the influence of the morphia.

contact with the parietal membrane, adhesions form, the intrapleural space is obliterated and the granulomata are then able to complete their function and undergo sound cicatrisation (see Figs. 18 to 21 and Figs. 22 to 24).

The second type is less frequent ; it differs from the first in that the fibrosis is much more extensive and re-expansion of the lung is impracticable. There is, however, no ulceration. This implies that the granulomata are resisting the tubercle bacilli with difficulty, but not wholly in vain, and that with assistance, such as the improvement of the general power of resistance, the removal of the irritant fluid, and possibly the therapeutic effect of the oxygen, they may ultimately succeed. It will be found that after aspiration by oxygen replacement, repeated perhaps two or three times and without leaving too high a negative pressure (since in these cases the lung is incapable of expanding), the activity of the disease and the re-accumulation of the fluid will cease (Fig. 25). The difference in the health in such circumstances and in the working capacity of the patient are quite remarkable.

The third type is by far the most serious of the three and the prognosis is always grave. The lung is collapsed and both this organ and the chest wall are rendered immobile by the great thickening and fibrosis of the pleural membranes. The granulomata are in various stages of caseation, liquefaction and ulceration, and the base of the pleural cavity is filled with pus cells and debris.

Oxygen replacement is at most a palliative method of treatment, and has to be constantly repeated : the needle punctures may cause considerable intrapleural bleeding owing to the excessive granulation tissue formation. Pleurotomy with drainage, as an alternative to repeated aspiration, is inadvisable. It will relieve the mechanical disabilities due to the constantly re-accumulating fluid, but the opening in the chest wall will become infected by the tubercle, and secondary infection is certain to occur. Such an operation will result therefore in an intractable chronic empyema.

There is one line of treatment which may possibly meet with a measure of success. The pleural membranes cannot be brought into contact by the expansion of the lung, but it may be possible to effect this approximation by the mobilisation of the chest wall. This can be done either by complete decostalisation or by rib mobilisation :* the latter does not produce such complete falling in of the chest wall as the former and would therefore not be applicable to those cases where the lung is completely collapsed.

It must be fully realised that this type of tubercular pyothorax indicates failure of the granuloma, and that the probable cause of such a complete failure is the very low resistance of the patient to the invasion of the tubercle bacilli. It is obviously useless, therefore, to attempt such a serious operation if there is tubercular infection, however slight, in any other organ or part of the body.

*This operation is described in Chapter IX.

Attention must be called to one danger associated with aspiration of tubercular pleurisies when the degree of resistance is low. The intact pleural membrane is a very efficient barrier, as has already been stated, to the spread of the disease into the chest wall. The gap in the barrier caused by puncture of the parietal pleura with a large trocar and cannula may allow of the spread of infection into the tissues of the chest wall. The first indication that this has taken place is the appearance of a small subcutaneous abscess two or three weeks after the puncture. The skin overlying the pus ulcerates and a permanent sinus is established.

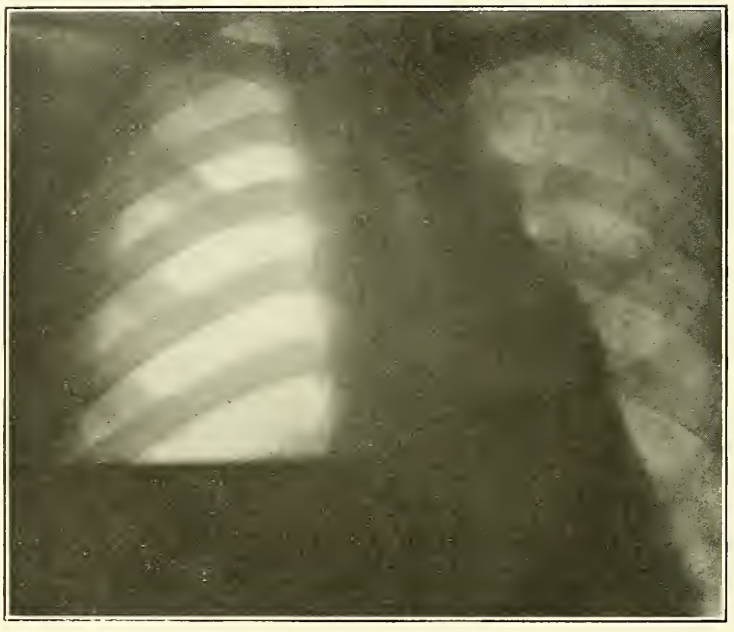

Fig. 22. Case F. R. August. 1913. Tubercular hydro-pneumothorax Right side. Before treatment by oxygen replacement.

Treatment of Tubercular Hydro- and Pyo-pneumothorax.

The principles determining the treatment of these conditions are precisely similar to those just described for tubercular effusions without the presence of gas. Very occasionally persistence of the opening of communication between the pleural cavity and the bronchus may necessitate special treatment (see below).

The satisfactory result obtained in a patient who suffered from tubercular hydro-pneumothorax of six months standing is shown in Figs. 22, 23 and 24. It is interesting to compare with this the case of another patient who had had a pyo-pneumothorax for three months only. This man had less power

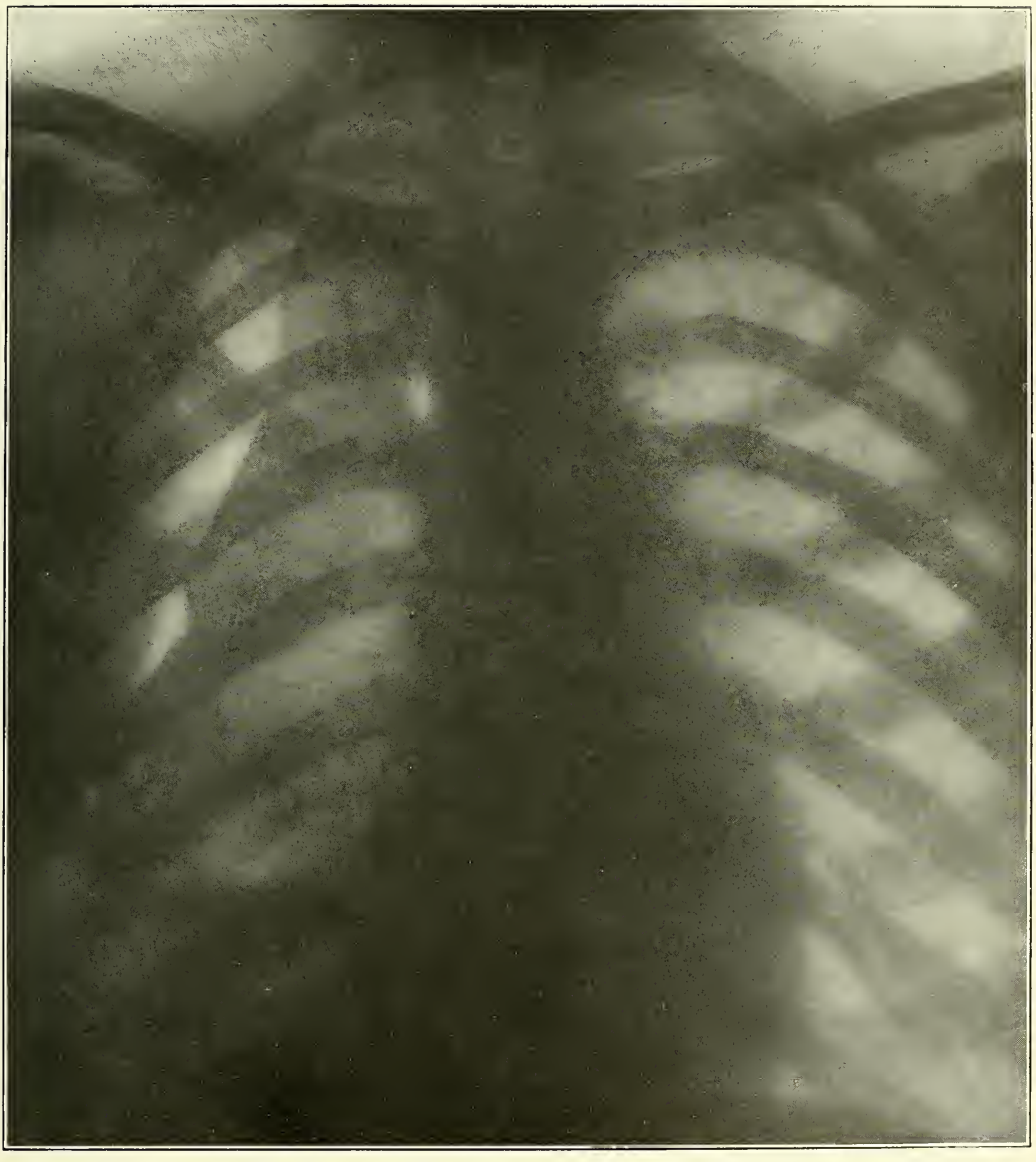

Fig. 23. Same case. September, 1913. Showing partial expansion of the lung.

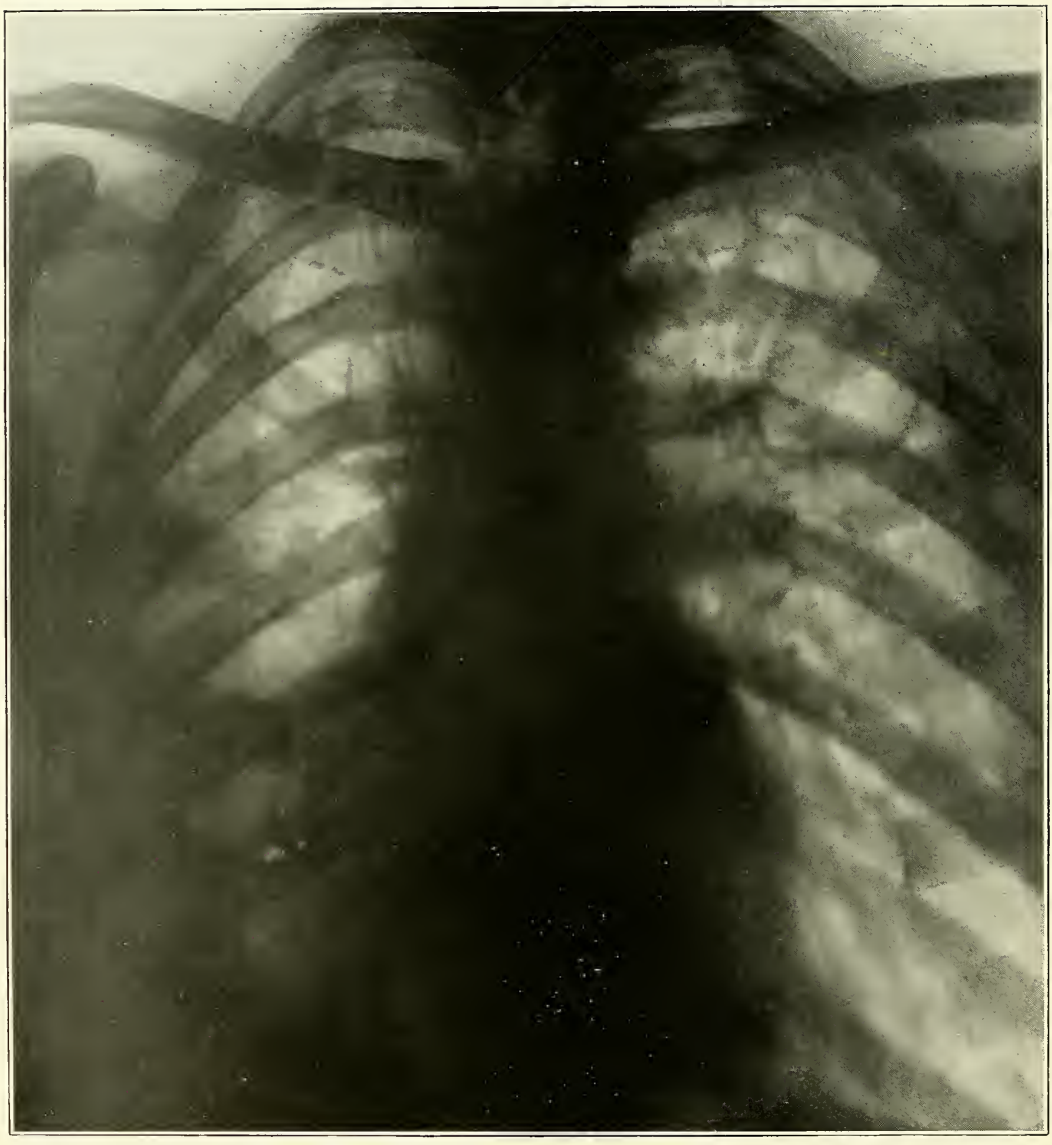

Fig. 24. Same case. February, 1914. Showing the lung expanded and the visceral and parietal membranes in complete apposition.

of resistance to the tubercular infection and it was quite impossible to produce any expansion of the lung or displacement of the mediastinum to the affected side. Treatment by oxygen replacement, however, gave him great relief, as it removed the weight of the fluid and prevented the recurrence of it. The skiagram illustrated in Fig. 25 was taken one month after the

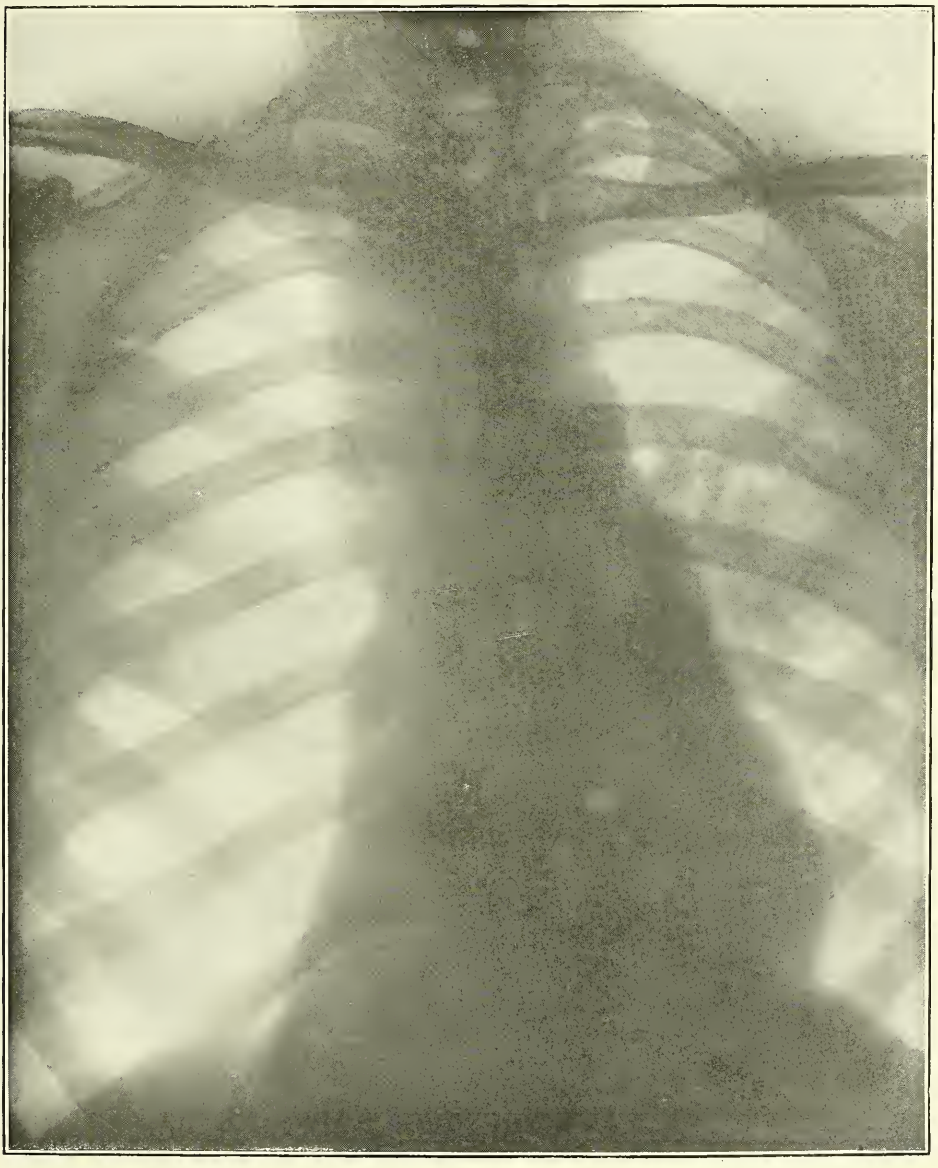

Fig. 25. Case T. H. Tubercular pyothorax. Right side, March 28th, 1912. Lung completely fixed by thickened pleura. Oxygen replacement had been done on the 21st and 29th of the previous month, 2,650 c.c. of fluid having been removed

end of the treatment. Whereas previous to the oxygen replacement he had had "the greatest difficulty in breathing on the least exertion" and had been wasting, he was able, six weeks after, to resume active work. He gained considerably in weight, and suffered from slight shortness of breath only.

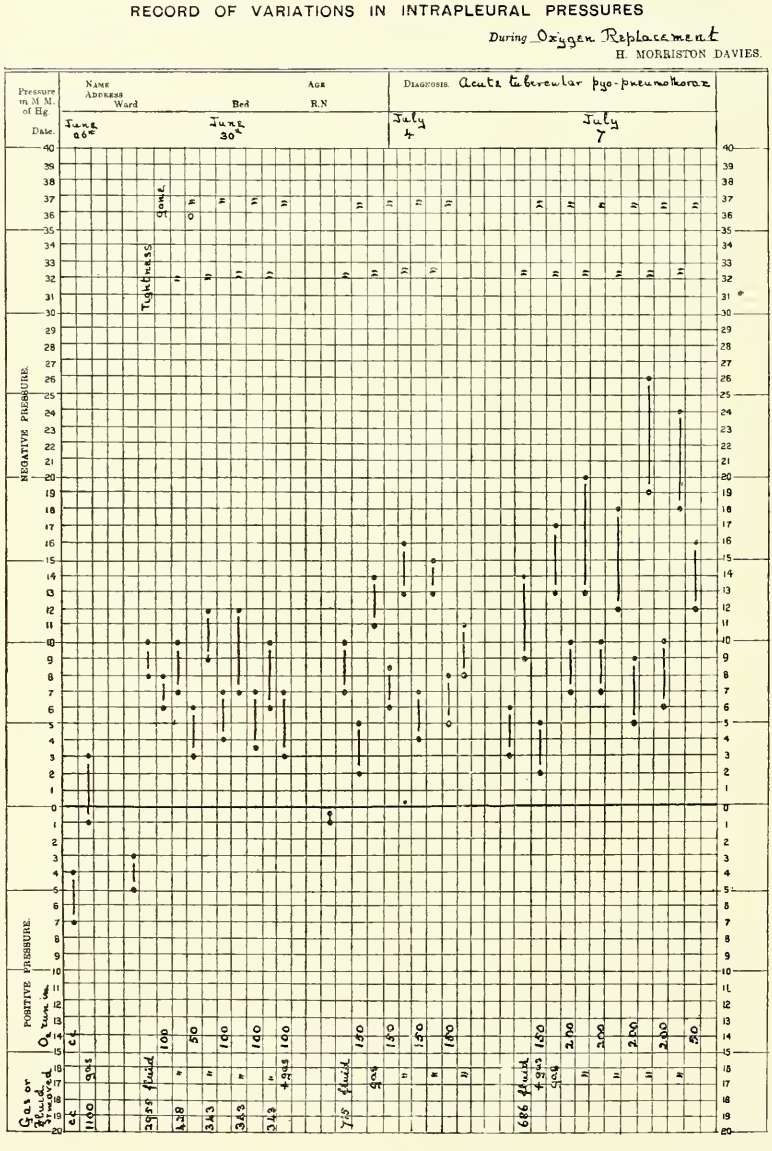

Fig. 26. - Case W. S. Acute tubercular pyo-pneumothorax. Chart showing variations in pressure during treatment of the case by oxygen replacement. On June 26th sufficient gas only was removed to relieve the symptoms, as it was not known if the opening in the lung had closed. The upper dots denote the inspiratory pressure. The lower dots denote the expiratory pressure.

CLOSURE OF THE OPENING IN THE LUNG.

Operative intervention for this purpose is rarely necessary except when the persistence of the opening is due to the inability of the lung to collapse completely owing to adhesions.* This was the state of affairs in the patient the skiagram of whose chest is shown in Fig. 27. An attempt directly to close the opening is most unlikely to meet with success owing to the great difficulty in finding it, but if the lung can be freed by dividing the adhesions,

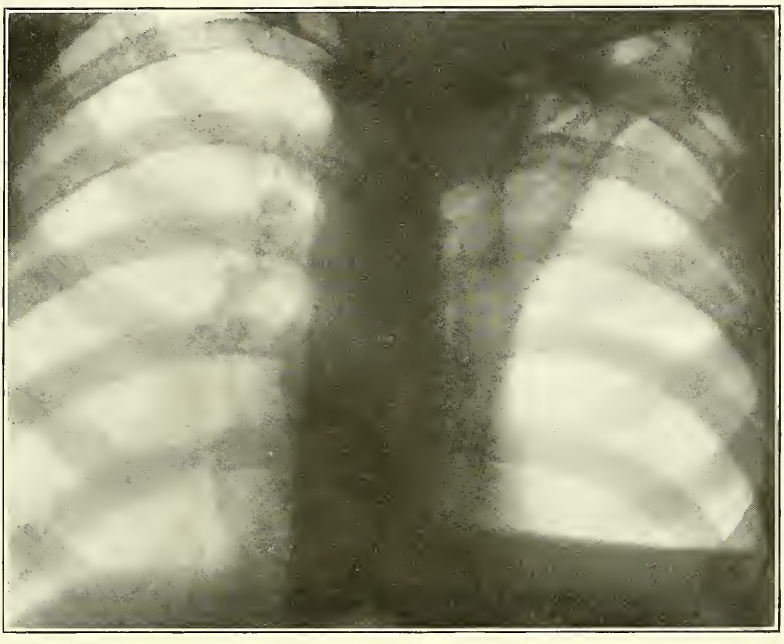

Fig. 27. Case C. C. Pyo-pneumothorax, left side, with communication between the lung and the pleura. As soon as the adhesion was divided, the lung collapsed and the opening became obliterated.

closure of the opening may result.† When the adhesions consist of isolated strands of stretched fibrous tissue, these can be divided either by a tenotome passed through an intercostal space and guided by indirect vision‡ or by obtaining direct exposure through an incision in the chest wall.

When dividing a band of adhesions by the open method, the incision through the chest wall should be made as close to the adhesion as possible and should be from 10 to 15 cm. long. The adhesion is cut through with a

* It is surprising how long, sometimes, a tubercular pyo-pneumothorax may communicate with a bronchus through an opening in the lung without becoming secondarily infected by pyogenic organisms.

† In the case referred to immediately above (skiagram Fig. 27), spontaneous closure followed on the division of the dense adhesion which can be seen in the skiagram passing upwards and outwards from the lung across the upper end of the pneumothorax.

‡ This procedure is described more fully in the discussion on the division of adhesions in cases of phthisis (see Chapter VIII).

knife, and if the opening in the lung is seen, the margins of it are approximated with catgut sutures. The bleeding from the cut edges of the adhesion is rarely sufficient to cause any anxiety. The technique of the incision through the chest wall and of the closure of the wound is precisely similar to that which has already been described in connection with the removal by pleurotomy of fibrinous effusions (see pp. 59 and 60). In the after treatment, the same care also is required to prevent the occurrence of a positive pressure and the accumulation of an effusion. The high negative pressure recommended after pleurotomy for the removal of an effusion is inadvisable, however, after pleurotomy for the closure of a perforation of the lung, until it is certain that that opening is soundly healed. During the first ten days or so, every effort should be made to keep the intrapleural pressure slightly below atmospheric.

Pyothorax (Empyema).

The clinical manifestations of this disease have already been discussed in the sections dealing with effusions, but there are two difficulties in connection with the diagnosis that may well be drawn attention to again for the sake of emphasis.

The differentiation of a purulent from a simple effusion except by exploratory puncture is, in most cases, impossible. There is often quite wrongly a prejudice against an exploratory puncture, and all too frequently therefore a pyothorax has been left untreated for weeks in the hope that the condition is a simple effusion. By the time that the lesion is recognised and treated, the lung has become fixed down by adhesions and thickening of the pleura, and is incapable of expansion. This is the cause of at least half the cases of chronic discharging empyema.

It is not uncommon for a pyothorax, developing during the course of an acute disease (*e.g.*, pneumonia), to produce a slight exacerbation only of the existing symptoms. Subsequently the acuteness of the condition may subside, the patient improves and the persistence of dullness is attributed to thickened pleura or imperfect resolution of the pneumonia. The empyema has become *latent*; this means not so much that there are no symptoms, but that they are insufficient to arouse suspicion as to the nature of affairs.*

A pleural effusion having been recognised, the possibility that it is purulent should always be considered, but more particularly so, when the patient is a child ;† when the effusion is secondary to some disease especially

* The patient's convalescence is, as a rule, slow and incomplete. He, in other words, "does not regain his old strength and energy." In children there may be chronic irritation of the upper end of the psoas muscle resulting in chronic contraction of the muscle, slight flexion of the thigh and limping.

† An effusion in a child under the age of five is usually purulent; under the age of three it is always so.

liable to be complicated by pyothorax, such as rupture of a tubercular cavity of the lung, pneumonia or appendix abscess ; when the effusion is initiated by a rigor or vomiting, or when there is an exacerbation of the symptoms and the development of a swinging temperature in pneumonia.

Cases of pyothorax may for the purposes of treatment be separated into four groups :—

(1) Those due to the tubercle bacillus only ; these have been discussed in the preceding pages.

(2) Those due to the pneumococcus only.

(3) Those due to pyogenic organisms, such as streptococcus, staphylococcus and bacillus coli, whether the infection is pure or mixed.

(4) Pyogenic infections in children under two years of age.

Before a line of treatment in a case of pyothorax can be determined, the fluid drawn off at the exploratory puncture must always be examined to ascertain the nature of the infection.*

TREATMENT OF PYOTHORAX DUE TO THE PNEUMOCOCCUS.

Pneumococcal infections vary greatly. The effusion may be clear, or it may be opaque from the number of cells or the amount of fibrin. The absorption of toxins may be so slight as to produce no more symptoms than a simple pleural effusion, but, on the other hand, it may be so great as to resemble in its clinical picture a pyogenic infection.

Aspiration by oxygen replacement is the first line of treatment to be adopted whatever the character of the effusion and whatever the clinical manifestations. Failure to remove the fluid owing to the fibrinous contents is an indication for pleurotomy followed by immediate suture of the wound, as detailed on pp. 59 and 60. In the toxic cases, the symptoms will usually yield temporarily to either the one or the other of these methods, but will probably recur in the course of the next three or four days. The temperature should be taken as the main guide. If the return of febrile symptoms can be immediately checked by oxygen replacement of the re-accumulated fluid, and the temperature remains approximately normal for an increasing period of time after each aspiration, this method of treating the pyothorax can be continued and a completely satisfactory result will in all probability be obtained in a shorter time and with less risk to the patient than by an open operation and drainage. Should, however, aspiration with oxygen replacement, independently of or as an accessory to pleurotomy with immediate closure of the wound, fail to check the processes of toxic absorption, the pleural cavity must be opened and drained without delay ; the drainage tube, in the absence of secondary infection, need not and should not be left in for more than three or four days.

* Sterility of the culture suggests that the pyothorax is tubercular.

TREATMENT OF EMPYEMA DUE TO PYOGENIC INFECTIONS.*

There is only one possible method of dealing efficiently with these cases and that is by incision, rib resection and free drainage. A regional or local anæsthesia, combined with morphia, is preferable to a general. If means for giving the former are not at hand, chloroform should be used in preference to ether. It is always advisable before operating on a large empyema under a general anæsthetic to aspirate from 500 to 1,000 c.c. of the fluid so as to abolish the positive pressure in the pleural cavity.

The patient when placed on the table must never be laid on his sound side, as the heart will be embarrassed and the working lung compressed between the table on the one side and the weight of the fluid on the other. The patient must rest either on his back in a semi-recumbent posture close to the edge of the table, or on the affected side. The former is the position for the axillary incision and the latter for the dorsal.

The opening into the chest must be as low as practicable and is usually made by removing the 9th rib in the scapular line, or the 8th rib in the mid-axillary line. If the opening is at a lower level, it is liable to become obstructed by the diaphragm. The incision may be either vertical or oblique along the line of the rib. The former has the advantages that it is easily extended downwards for removal of part of a lower rib if the original opening is too high, that in the dorsal incision the fibres of the latissimus dorsi muscle are split and not cut, and that at the end of the operation it is easy to ensure that the opening in the skin is opposite the opening in the pleura. The oblique incision needs a smaller area of local or regional anæsthetisation ; but there is always the risk that the opening through the skin will not correspond with that through the deeper tissues.† I prefer the axillary route because it is easier to have the patient on his back during the operation ; the scapula and latissimus dorsi muscle are not in the way and there is less tissue to divide ; the space between the ribs is greater and the drainage is equally efficient. The incision should extend from 4 cm. above to 4 cm. below the centre of the portion of rib to be removed. The tissues are retracted,‡ the rib exposed, stripped of its periosteum, and a length at least 5 cm. long excised.‖ The periosteum and parietal pleura are divided along the axis of the rib, and as the pus escapes the gloved finger is at once inserted into

* Tubercular and pneumococcal infections are not included in this group.

† As long as upward movement of the arm is confined to the shoulder joint there is no movement of the skin on the thorax ; but when the scapula comes into play, the skin over the chest is drawn upwards.

‡ All manipulation must be done with great care and deliberation when a local anæsthetic is used. The patient should always be warned before the bone is cut across that he will feel a painless jar.

‖ I always inject the intercostal nerves on the proximal side of the divided rib with a few minims of absolute alcohol. This does away with the after pain caused by the cut end of the ribs irritating the pleura.

Bleeding from the intercostal artery owing to damage when dividing the rib may be difficult to control unless either a further portion of the rib is removed or a ligature applied right round the rib including the vessel.

the opening to control the rate of flow and prevent too sudden a return of the displaced mediastinum. The finger is able at the same time to explore the lower part of the cavity, to break down the walls of any loculi, and to gauge the capacity of the lung for re-expansion. A drainage tube, at least 2 cm. in diameter, is inserted into the wound ; it should project at most 2 cm. beyond the parietal pleura and should not touch the diaphragm.* A large safety pin is passed through the outer end of it to prevent it slipping into the cavity.† The two extremities of the cutaneous part of the wound are stitched up and the whole is covered with a very large absorbent dressing.

If it is found that the opening into the pleura is too high or two small, part of the rib below must be removed. There is no disadvantage in making the opening too large ; there are many in making it too small.

After Treatment.

The essentials of this are the maintenance of free drainage, early removal of the tube, the prevention of re-infection and the encouragement of the lung to re-expand.

Free drainage is of the utmost importance since, if there is retention of pus, the organisms become increasingly virulent, there is a greater danger of secondary infection and a further deposit of fibrin on the walls of the cavity will take place ; the capacity for re-expansion of the lung will thus be greatly diminished.

The early removal of the tube is important because the tube acts as an irritant to the tissues and may lead to the formation of a chronic sinus, caries of the adjacent rib, or even secondary hæmorrhage from the artery.‡ A tube leading from the skin into a serous cavity is moreover always a possible source of secondary infection. As soon as the discharge diminishes in amount and becomes serous in character, the tube is omitted and if a sufficiently large opening was made at the operation, this is not likely to close before the empyema is healed. A tendency to too early closure of the external wound can be checked by the occasional replacement of a tube for twenty-four or forty-eight hours.

Irrigation of the wound with liquids is inadvisable owing to the dangers of pleural reflex (see Chapter V). In a very offensive empyema, a slow stream of ozone may be allowed to escape into the cavity through a catheter, but it is most essential that the opening round the catheter for the escape of the gas should be considerably larger than the diameter of the inlet tube.

The re-infection of the wound can be guarded against only by careful dressings and daily removal and sterilisation of the tube, and by keeping the skin which is in contact with the tube sterile by the use of some antiseptic

* Pain and a distressing cough may be caused by irritation of the diaphragm by a tube.

† Tubes are made with external, or external and internal flanges, but they have no special advantages.

‡ This last is rare as the vessels have usually become thrombosed.

lotion. Patients are too frequently sent out of hospitals or nursing homes before the empyema cavity is completely obliterated; they become re-infected and develop a chronic sinus.

Innumerable varieties of artificial means to encourage expansion of the lung have been tried. The appliances which can be attached over the wound and exert a continuous suction on the contents of the cavity, including the lung, are of comparatively little use and tend to interfere with the movements and comfort of the patient. When the disease is diagnosed and treated sufficiently early, there is no need for any of these appliances; they are not powerful enough when the operation has been delayed. Breathing* and physical exercises should be encouraged as soon as the patient is well enough.

TREATMENT BY SYPHON DRAINAGE.

Children under two years of age tolerate very badly the ordinary operations for empyema. As an alternative measure and as one giving more satisfactory results, Holt† recommends that empyemata in infants should be treated by syphon drainage. The principle is as follows :‡ "Exploratory puncture is made at the intended site of operation with the needle, which is allowed to remain in as a guide. A puncture incision is made with a bistoury or small scalpel, . . . The wound should be a little smaller than the drainage tube to be used as it can be readily stretched in introducing the tube. The drainage tube should be firm and of large caliber (18 F) . . . Before insertion there is cut from the end of the drainage tube a small ring, a little more than one-eighth of an inch wide. This is slipped over the end of the tube to a distance of about an inch and forms a collar to prevent the tube slipping into the chest cavity. Just above this is placed a piece of broad tape with a small buttonhole in its centre, which fits snugly against the drainage tube and pulls against the collar formed by the rubber tube previously mentioned. The tube is inserted into the chest up to the collar or about an inch; the tape is then applied against the chest wall, and is held in place by four or five strips of adhesive plaster."‖ These hold the drain in position and make the opening airtight. The drain is joined by a glass connection to a length of rubber tubing, the distal end of which is attached to a glass tube passing through a cork to near the bottom of a bottle, with a capacity of at least one pint. This bottle contains sterilised saline solution. The saline must be changed frequently during the next few days, the rubber tubing being meanwhile clamped.

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* *e.g.*, blowing up of an air cushion or blowing water from one Wolff's bottle to another.

† "The Syphon Treatment of Empyema in Infants and Young Children Compared with other Measures." *Americ. Med.*, Vol. 19, page 381. 1913.

‡ The technique is a modification of that introduced by Bulau.

‖ Holt. "Syphon Treatment of Empyema." *Americ. Med.* 1913.

Apical, Diaphragmatic* and Interlobar Empyema.

To the ordinary difficulties of diagnosis of the existence of a pyothorax are added the unexpectedness of the situation in an apical empyema, the depth of the pus, the presence of lung tissue between it and the wall, and the absence of signs of fluid in the diaphragmatic and interlobar varieties. Apical empyema must be suspected when signs of fluid at the apex appear during the course of an apical pneumonia or phthisis. Delay of resolution and the onset of toxæmic symptoms in a basal pneumonia, especially when there are localised signs of compressed lung tissue, suggest diaphragmatic empyema. A basal empyema may give rise to an irritating cough and even vomiting. The X-rays will always reveal the localised collection of fluid, the nature of which can be determined by an exploring syringe and an especially long needle.

The treatment of an apical empyema calls for no special mention. The opening should be made through the front of the chest wall unless the cavity extends sufficiently low to be reached through the axilla.

In opening a diaphragmatic empyema care must be taken that the pleural cavity (if such exists) above the adhesions is not opened.

An interlobar empyema must be treated with the same respect and in the same manner as a pulmonary abscess (*q.v.*). Such a collection of pus is particularly apt to invade the lung tissue and to rupture into a bronchus.

Bilateral Empyema.

The treatment of bilateral empyema is based on the same principles which underlie that of unilateral empyema, with this exception, that the radical operation on the less affected side must be temporarily delayed. The side on which there is the greater amount of lung collapse requires immediate attention—aspiration or pleurotomy with oxygen replacement if the condition is due to pneumococcus ; free opening and drainage when the pus is caused by streptococcus, staphylococcus or mixed infection ; syphon drainage in infants. On the next day (or the same if the patient can tolerate it) the pus in the opposite pleural cavity should be removed by aspiration with oxygen replacement ; in infants, syphon drainage on that side also should be instituted. When drainage of one cavity has been established and the other side fails to clear up under the less energetic treatment, drainage may be established of that cavity also, provided, however, that the lung on the side first operated on is expanding satisfactorily. The one wound must be always well protected by dressings while the tube on the other side is being changed.

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* The term is here used to denote collections of pus completely enclosed between lung and diaphragm.

Spontaneous Escape; Pointing Empyema.

The pus from an empyema may escape by perforating into a bronchus or through the chest wall. Rupture into a bronchus may result in the complete evacuation of the pus and in a complete cure. This successful termination is rare except in the case of a small localised collection of pus. Rupture of a large empyema through a bronchus may cause death by suffocation. When the patient survives, the cavity becomes re-infected through the bronchus and a pleuro-bronchial fistula results. Operative treatment may be delayed only when there is every indication clinically and radiologically that the drainage through the bronchus is efficient, and that the cavity is becoming obliterated by the approximation of the surrounding walls.

Escape of the pus through the chest wall is seen most frequently in children and young adults. Ulceration of the parietal pleura is followed by leakage into the tissues of the chest wall, the formation of a subcutaneous abscess (pointing empyema; empyema necessitatis) and eventually of a fistula. Such a perforation occurs usually in the front of the chest through the upper intercostal spaces (Fig. 28) or through the 5th or 6th space in the axilla.

Pulsation may be observed in the subcutaneous tumour of a pointing empyema. It occurs most frequently in association with large left-sided effusions and is due to the transmission of the heart's impulse.* It depends on the opening in the chest wall being large and direct—conditions that will also allow transmission of an impulse when the patient coughs.

COMPLICATIONS.

Rupture of an empyema into a bronchus or through the chest wall has been discussed in the preceding section. The pus may find its way into the lung parenchyma and ooze through into a bronchus, giving rise to purulent expectoration with the symptoms and possibly signs of a lung abscess. More rarely there is invasion of the psoas sheath and the formation of a psoas abscess; or there may be perforation through the diaphragm or into the œsophagus.

Gangrene of the lung is due to rupture of an empyema into the lung parenchyma. Gangrene and sloughing of the surface is found only when the infection is extremely virulent; such an infection may also produce an acute cellulitis or a diffuse abscess of the chest wall.

Cerebral abscess is a complication of chronic rather than of acute empyema, and is particularly liable to develop after operations on chronic cavities (see Chap. IX).

* Two varieties are met with in association with pleural effusions, the one is of the character of a thrill felt over a wide area, the other is the pulsation observed in association with the local swelling. They are observed most commonly in cases of purulent effusions (with or without air), but sometimes also with serous or hæmorrhagic effusions.

The fluid in all cases of hydro- or pyo-pneumothorax with complete collapse of the lung can be seen with the X-rays to pulsate synchronously with the heart's action.

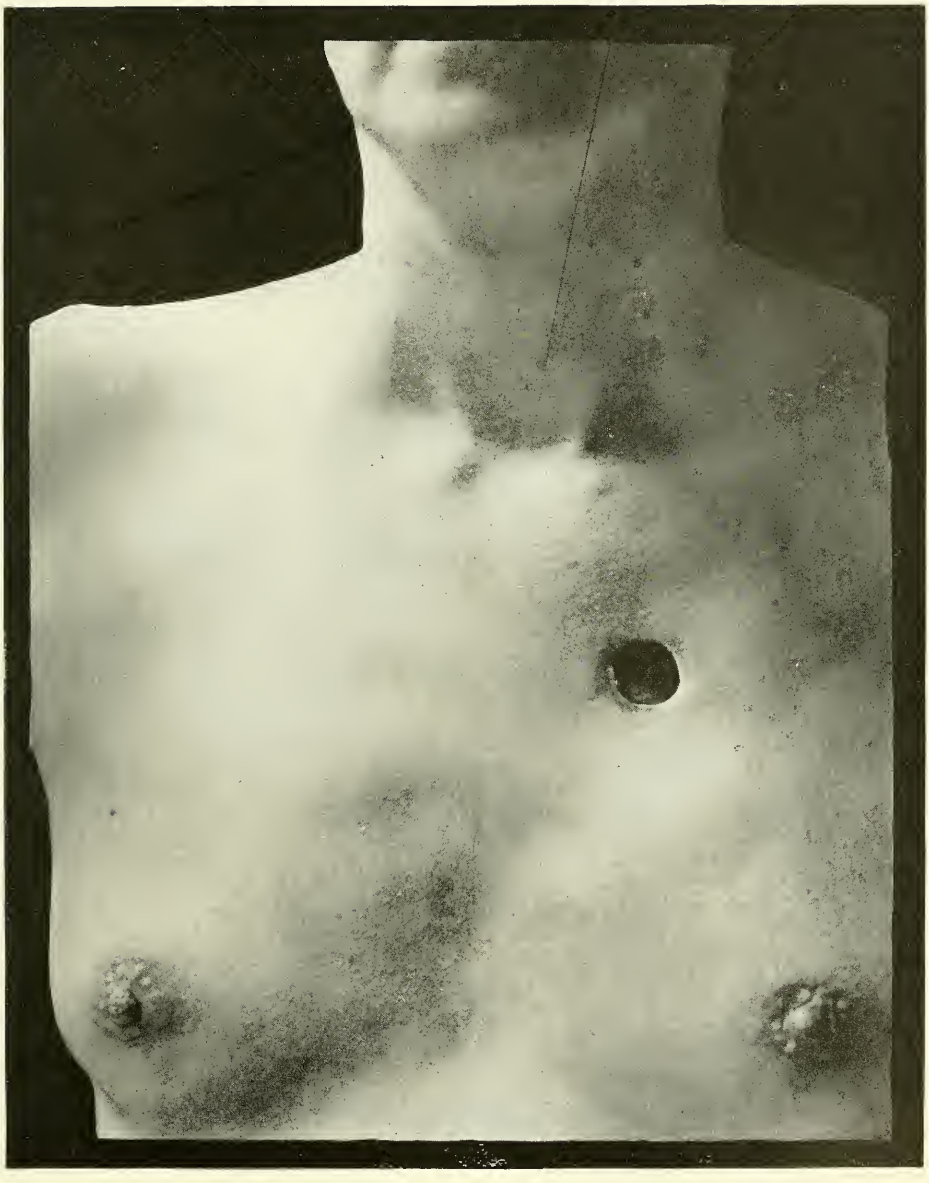

Fig. 28. Photograph showing the opening of an empyema which has ruptured spontaneously.

Pericarditis, with or without effusion, may develop as a result of the irritation caused by the adjacent pus especially in cases of left-sided empyema.

Calcification of the pleura. Occasionally there is found, attached to the parietes, or to the diaphragm, or forming a hood closely investing the apex of the lung, a densely thickened mass of fibrous tissue, embedded in which is a single irregular plate, or a series of plates or stalactites, irregular as to outline, surface and thickness.* Between these calcareous masses and the fibrous tissue there is sometimes a mass of debris and caseous material. There is little doubt that calcification is a sequela of chronic pleurisies, particularly of small empyemata which have remained untreated and which have become gradually, in part, absorbed.

The interference with the free movements of the lung and chest wall causes shortness of breath. The symptom may not be sufficiently marked to attract special attention (it is latent) or it may be embarrassing and progressive.† The signs are those of chronic pleurisy ; they are not, however, uniformly distributed, but are increased in degree over the areas of calcification. The dense irregular shadow seen with the X-rays is usually diagnostic.

Unless interfering with the patient's activity, the condition is best left untreated. If radical treatment is adopted, it is necessary that the whole of the calcareous deposit should be removed, as a chronic sinus and suppuration almost invariably follows a partial operation.

Access to the affected part is obtained by a long intercostal incision (see p. 237), and the thickened fibrous tissue, including the plates, is stripped off the parietes or off the surface of the lung as in the operation of decortication (see p. 79).

Chronic Discharging Empyema.

The morbid anatomy of this condition has been already sufficiently indicated in the preceding pages.

The causative factors of chronic empyema are :—

Delayed operation.

Insufficient drainage ; due to too small or to too rapid closure of the opening, or to its being too high.

Foreign bodies in the pleural cavity, *e.g.*, drainage tube, gauze, rib sequestrum or metal fragment.

The character of the infection, *e.g.*, tubercle, streptothrix or re-infection.

Bronchial fistula.

* They are composed mainly of calcium phosphate, some calcium carbonate, magnesium phosphate and calcium fluoride.

† Spontaneous suppuration may be the first indication of the condition.

A consideration of this table can lead to one conclusion only, namely, that practically every case of chronic empyema is due either to a mistake in diagnosis or to a mistake in treatment. In other words, 99 per cent. of chronic empyemata are preventable.

Empyema due to a streptothrix infection is considered in Chapter X.

A chronic tubercular pyothorax or an empyema which involves practically the whole of the pleural cavity is difficult to cure. All other cases of empyema can be healed, provided that sufficiently energetic measures are adopted.

TREATMENT.

An empyema cavity which has failed to close within three months of the operation for drainage must be regarded as a chronic empyema and in need of further intervention.

Whatever the cause or the size of the chronic cavity, the principle of the treatment is the same and consists in bringing the walls of the space into complete contact. Any operation which fails to accomplish this fails to produce a cure. The technique varies with the size and shape of the cavity.

TREATMENT OF A SMALL FLAT CAVITY.

(Capacity not more than 200 c.c.)

In this type of case the walls are nearly, but not quite, in apposition ; the drainage opening is often above the lower level of the cavity and is narrow by reason of the new growth of bone. The opening may be an oblique tract owing to the ascent of the diaphragm. There may be a foreign body. Healing can in all probability be obtained by providing efficient drainage, by removing the overlying ribs, and by extracting the foreign body, when present.

A vertical incision, unless some other direction is indicated by the position of the wound or the shape of the cavity, affords the best access. The whole of that part of the length of every rib overlying the cavity (and a little more besides) is stripped of periosteum and removed. The cavity is explored by enlarging the opening into it, and all debris and foreign body (if any) removed. A large drainage tube is inserted into the extreme lower end of the space and the rest of the wound is closed.* The tube is removed, sterilised and replaced on the third and every succeeding day until the tenth, when, as a rule, it can be dispensed with.

If the cavity does not close after this operation, it must be treated by the method described below.

* This method is similar to that originally described by Estlander; he, however, advocated a series of incisions parallel to and between two adjacent ribs giving access to the one above and below.

TREATMENT OF CHRONIC CAVITIES OF MODERATE SIZE.

(Capacity between 200 and 1,000 c.c.)

As a general rule, the cavity is between the lung and the axillary part of the chest and is roughly wedge-shaped with the base below.

There is considerable danger that, owing to the very thick and dense fibrous walls of the abscess cavity, the patient may have lost a great deal of his acquired immunity to the infection and that the disturbance of the parts may produce temporarily a condition of acute infection. If a large area of raw tissue, such as is produced by the necessary operative measures, is exposed to the infection, the absorption of it may lead to fatal results. It is always advisable, therefore, to do the operation in two stages ; at the first, opening up the cavity so as to obtain free drainage ; at the second, mobilising the wall of the cavity.

First stage. The mass of new bone which has formed round the drainage opening is removed through an oblique incision parallel to the ribs. The cavity is explored, all debris and any foreign body is removed and a large drainage tube inserted. If the original opening is placed too high, a vertical incision must be made downwards and a portion of the rib, or of one or two ribs, below removed. The after treatment is the same as for acute empyema.

Second stage. This is undertaken after an interval of ten days at least ; if there has been a rise of temperature, the operation is postponed until five days or more after the return of the temperature to the normal or to its original level. A vertical incision is made extending up the mid-axillary line* from the base of the cavity to a distance of one rib above the upper level of the empyema cavity, when such is possible.† Across the lower end of the incision a second one is made parallel with the ribs and with a slight upward curve at either end. One or other of these cuts should be planned so as to extend into or traverse the drainage opening. The soft tissues are stripped off the bony framework, and that part of each rib which overlies the cavity and at least 1 cm. more in length on either side is denuded of periosteum and removed. Part of the rib above can also be removed with advantage. It is wiser to remove too much than too little bone. The outer wall of the cavity now consists of thickened pleura, periosteum and intercostal muscles, including the vessels and nerves. The whole of this is cut away, the knife being carried along the posterior border first, so that if any of the vessels are not obliterated, they shall only bleed the once when cut. The two flaps of soft tissue are replaced and stitched together, leaving, however, a large opening for drainage at the lower extremity of the cavity. These flaps should become firmly adherent to the underlying lung, and the whole cavity be thus oblitrated, the drainage wound closing by granulation.‡

* The position of this incision must, of course, be altered to suit a cavity localised on the anterior or posterior aspect of the chest.

† The axillary incision gives access for the removal of ribs up to and including the second. If the cavity extends up to the first rib, as much of this rib as possible should be excised through an anterior incision along the lower border of the clavicle, the arm being supported at right angles to the chest and the greatest care being taken to avoid injury to the axillary vessels.

‡ The operation just described is often erroneously referred to as Estlander's. It was Schede, however, who first appreciated the importance of removing the thickened parietal pleura. No operation on any but the smallest cavity can be successful unless a *very free* removal of the overlying and adjacent ribs and pleura is done.

TREATMENT OF LARGE EMPYEMA CAVITIES.
(Over 1,000 c.c.)

The difficulty in the treatment of these cases is due to the necessity of removing practically the whole of the ribs on the affected side, if the chest wall is to be brought in apposition with the lung. It is impossible to obtain access for the free removal of the upper ribs unless all the muscles passing from the scapula to the chest are divided, and that bone is dislocated upwards with a large musculo-cutaneous flap. The amount of tissue which must be removed for the operation to be a success is the whole of that part of every rib, of the intercostal muscles and of the parietal pleura which overlie the cavity. (Schede's operation.)

Technique.

First stage. Preliminary to the main operation, the local rib removal and free drainage described already on p. 78 are essential.

Second stage. The original incision, slightly modified, advocated by Schede is the most suitable. The incision begins on the front of the chest over the pectoralis major in the mid-clavicular line opposite the 3rd rib, extends downwards to the 10th rib in the mid-axillary line, curves then backwards and upwards running parallel to, and one cm. internal to the vertical border of the scapula, as high as the 2nd rib posteriorly. The flap thus marked out should include all the tissues down to the chest wall except the pectoralis muscle anteriorly, which is not divided. When the flap is turned up and the arm is displaced upwards and forwards across the face the whole of the axillary part of the anterior and posterior surfaces of the ribs are exposed. These are rapidly denuded of periosteum by Doyen's elevator as far as the cartilages in front and the angles behind. The ribs are divided along the middle, the halves drawn asunder and broken off ; in front, the point of breakage will be the costo-chondral junction, behind, at the level of the tubercle.* The knife is then inserted into the pleural opening and the whole of the soft tissues overlying the cavity are cut right away. The flap is replaced and the wound closed† except at the lower angle through which a large drainage tube is inserted.

This operation is a very serious one and must not be undertaken if the patient is exhausted by chronic toxic absorption.

In certain cases it is possible, as an alternative measure, partially to *mobilise the lung* ; the mobilisation of the chest wall can then be proportionately less. A healthy lung is always capable of re-expansion, however long it may have been kept in a state of collapse. This fact is taken advantage of in the operation of *decortication* introduced by Delorme

* The ribs usually removed are the 2nd to the 10th inclusive. The first rib can be reached if necessary by prolonging upwards the posterior incision.

† The divided scapulo-thoracic muscles must be united by catgut sutures.

and Fowler. A longitudinal incision is made through the thickened visceral pleura, and is deepened gradually until the blue surface of the lung appears. It will be found now that the fibrinous deposit can be peeled in sheets off the surface of the lung, and as this constricting sheath is removed the lung expands. Care must be taken not to tear the lung tissue, and special attention must be paid to the removal of the fibrinous deposit along the lines where it passes from the surface of the lung to the chest wall.

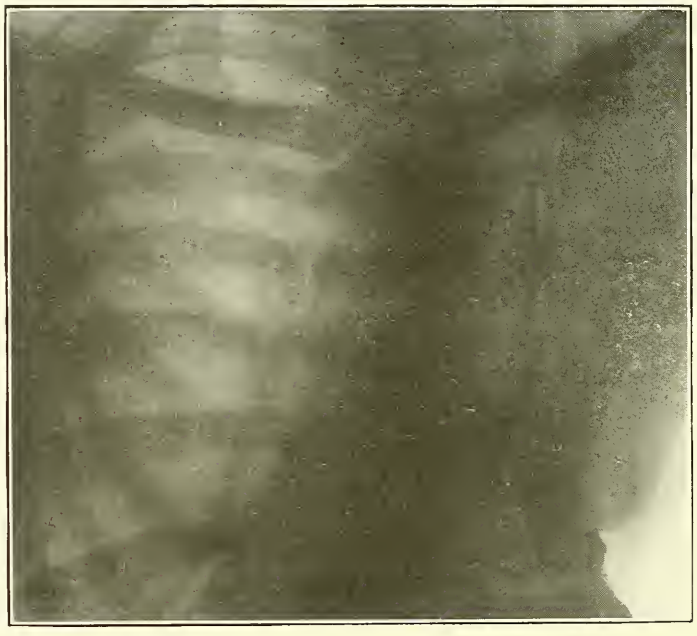

Fig. 29. Skiagram showing the appearance of the chest after Schede's operation.

Ransohoff found that a similar beneficial effect could be produced by a series of vertical and horizontal incisions through the thickened pleura a third of an inch apart. This operation is known as *discission*.

TREATMENT OF CHRONIC SINUS.

These sinuses through the chest wall end usually in a slight dilatation in the region of the pleura. The dilated extremity is lined with septic granulation tissue and contains possibly a fragment of necrosed bone or a foreign body such as gauze. Healing will sometimes follow the removal or spontaneous evacuation of the foreign body, but as a general rule, the complete excision of the track is necessary, and special care must be taken that the whole of the lining of the distal extremity is removed, together with any foreign body that may exist. When the track is hemmed in by the

formation of new ribs, the surrounding bone must be freely excised. The wound should be lightly packed with gauze soaked in saline solution, and allowed to granulate from the bottom.

Occasionally a sinus is due to the persistent use of a drainage tube, abolition of which is then in itself sufficient treatment.

TREATMENT OF BRONCHIAL FISTULA.

The opening from the bronchus may communicate with a large empyema cavity, or, when the lung is expanded and adherent, with a fistula in the chest wall. In the former case, the empyema cavity must be treated as described in the preceding pages, and the bronchial fistula closed by excising the margins, approximating the walls by a catgut purse-string suture and by a second row of Lembert sutures.

When the bronchial fistula communicates directly with a sinus, the fistula should be excised, together with the walls of the sinus, and the whole wound packed lightly with gauze so as to obtain healing by granulation from the bottom.

Pneumothorax.

Gas in the pleural cavity is the result of :—

1. Rupture of a diseased lung. Phthisis is responsible for the great majority of the cases. It is occasionally a complication of gangrene and abscess, and, rarely, of bronchiectasis and emphysema. In this group also may be included pyo-pneumothorax due to rupture of an empyema into the lung.
2. Injury to the chest wall, bronchus or lung.*
3. Gases injected as a therapeutic measure.†
4. Operations which entail the opening of the pleura in the absence of general adhesions.‡

Gas in the pleural cavity uncomplicated by the presence of fluid is rarely found except in some cases of injury, and when pneumothorax is artificially produced. In practically all cases of rupture of the lung due to disease the condition is one of hydro- or pyo-pneumothorax ; the fluid may be hæmorrhagic. The pus is due to the escape, with the gas, of infective material from the ruptured cavity of the lung, whilst the serous fluid is an indication, probably not so much of simple irritation, as of a very mild infection.

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* This condition is dealt with also in Chapter VI.

† The use of oxygen has already been discussed on pp. 46 *et seq.* Nitrogen pneumothorax is dealt with in Chapter VIII.

‡ For the treatment of air enclosed in the pleural cavity after pleurotomy, see pp. 59 and 60. Open pneumothorax is discussed in Chapter II.

The gas exerts pressure equally on all the surfaces of the lung and on all the walls of the pleural cavity. Its initial effect is to produce collapse of the lung. As the intrapleural pressure is changed from negative to positive, it acts on the surrounding walls, which yield in proportion as they lack the power of resistance, *i.e.*, the mediastinum the most, the chest wall the least.

Theoretically, one would expect the lung, as it is displaced, to collapse on to the hilum in the form of a pedunculated rounded tumour, and, in fact, this does occur in cases of traumatic pneumothorax with a perfectly healthy lung (see Fig. 31). When the lung is diseased, however, its elasticity is interfered with, and in chronic lesions such as phthisis, the bronchi have become thickened and rigid. In such cases, the lung is collapsed as a flattened band into the vertebro-mediastinal sinus* (see Fig. 25). The lung, if free of adhesions, is never compressed into the apex as in cases of hydrothorax. The presence of fluid in conjunction with gas affects the displacement of the lung only in so far as the lower part of that organ is concerned.

SYMPTOMS AND SIGNS.

A pneumothorax produced under control is symptomless, but when due to the rupture of the lung produces the most acute manifestations. At the onset there is acute pain and the sensation of "something having given way." The shock to the patient is severe ;† dyspnœa is immediate and progressive and there is cyanosis. The embarrassment of the circulation owing to the shock, the rapid displacement of the heart and the deficient aeration may be fatal. The intensity of these symptoms depends on the size and character of the opening. If the aperture is free and the patient survives the initial shock, the symptoms will gradually subside. When, however, the opening is valve-like in character, allowing the escape of air into the pleural cavity but preventing its return, the pneumothorax is progressive and the intrapleural tension becomes increasingly positive (ingravescent pneumothorax). Sauerbruch‡ attributes this increasing pressure to the character of the breathing resulting from a closure of the glottis, which is a reflex condition due to the pain. The breathing is groaning and laboured. During expiration the air in the bronchi is forced into the collapsed lung, which it partially distends, and then through the opening into the pleural cavity. Coughing produces a similar change but in a greater degree.

In a complete pneumothorax, the chest on the affected side is expanded and motionless, the intercostal spaces are obliterated, the shoulder is raised,

* Occasionally, in cases of artificial pneumothorax, the lung can be seen with the X-rays as a flattened band attached to the mediastinum by the hilum, but with the upper part floating free from the mediastinum.

† The pulse is feeble and rapid, the expression is intensely anxious, the temperature falls and the extremities become cold.

‡ "Die Bedeutung des Mediastinal emphysems in der Pathologie des Spannungs-pneumothorax." *Bruns Beitrage*, Bd. 60, s. 450, 1908.

the diaphragm is depressed and the heart is displaced in proportion to the intrapleural pressure. The resistance on percussion is diminished—the finger feels to rebound off the chest wall—and the note is hyper-resonant and tympanitic in character. On auscultation the affected side of the chest is dumb, except close to the vertebræ where bronchial breathing over the compressed lung may be audible. The bell note may be obtained*: very occasionally a metallic tinkling is audible during inspiration, when there is free passage of air through an opening on the surface of the lung. The recrudescence of dyspnœa in a case of spontaneous pneumothorax is an indication usually of the formation of fluid; it may be due, however, to the re-opening of the rupture which has become closed by the pressure of the gas on the collapsed lung. In the former case the alterations in the physical signs are the dullness at the base of the cavity, the increase in the displacement of the heart and the splashing sound obtained when the patient is shaken (succussion sound). The upper level of the dullness is absolutely horizontal and remains so whatever the position of the patient (shifting dullness).

A pneumothorax is translucent to the X-rays, the outline of the heart, of the aorta (in left-sided pneumothorax) and of the compressed lung stand out sharply defined (Fig. 20). When fluid is present, the opacity of it with its horizontal upper border is in sharp contrast to the gas (Fig. 22). Pulsation of the upper level of the fluid, synchronous with the heart beats, is always visible on the left side and sometimes also on the right.

TREATMENT OF SPONTANEOUS PNEUMOTHORAX.

Treatment must be directed at first to combating the shock and to relieving the acute mental and physical distress of the patient. This is best accomplished by a subcutaneous injection of morphia, by giving oxygen and by placing the patient in that position in which breathing is least embarrassed; this is usually the semi-recumbent posture, reclining somewhat on the affected side. When the symptoms of distress are extremely urgent, immediate relief can be given by thrusting the point of a large hypodermic needle through the chest wall, thus allowing for the escape of some of the gas and a reduction of the high intrapleural pressure. This needle should be withdrawn as soon as the acute symptoms have subsided, lest the reduction of the pressure be so great as to delay or prevent the closure in the opening of the lung.

This opening in the lung may be a simple one allowing of the passage of air both in and out of the pleural cavity, or it may be valve-like, admitting air into the cavity but preventing its exit, thus producing a steadily increasing (ingravescent) pneumothorax. In either case, a positive intrapleural pressure when associated with *complete* collapse of the lung is almost certain

* The bell sound quality of the note heard with the stethoscope when tapping two coins together over an adjacent part of a pneumothorax, is by no means always present, but if heard is due to free gas or a large cavity in the lung.

to cause obliteration of the opening. When the collapse is incomplete owing to adhesions, the aperture may become closed if the intrapleural pressure is sufficiently high; but, on the other hand, the drag of the adhesions may be responsible for keeping the aperture patent.

It is obvious, therefore, that if the passage of air can be prevented by a positive pressure in the pleural cavity, it is very unwise so to reduce the pressure that air can escape from the ruptured lung. The sooner the margins of the opening are approximated and the more continuously the approximation is maintained, the sooner will the wound heal. Not infrequently the opening is quite small and the collapse of the lung that is obtained by the escape of air is sufficient in degree and in duration to produce a cure without any extraneous assistance. But when the opening remains patent as the result of the pull of adhesions, or it is valve-like in character, or is repeatedly re-opened by the violent movements associated with a cough which cannot be controlled, then surgical intervention is necessary for the purpose both of relieving the symptoms and of reducing the danger of secondary infection from the lung.

I have recently tried and since published a description of an apparatus which can be connected with a needle traversing the chest wall and which enables a positive intrapleural pressure of any desired degree to be obtained. The value of this is that it abolishes the distress and danger of an ingravescent pneumothorax or of a tear in the lung becoming suddenly re-opened during exertion or coughing. "If the needle which passes through the chest wall is connected by tubing to a Wolff's bottle part filled with water, the depth of the opening of the tubing below the surface of the fluid can be altered so that the gas will escape whenever the intrapleural pressure exceeds the required maximum, which should be equal to about 5 mm. of mercury. It will be found, however, that a cough raises the pressure considerably above this. With each cough, therefore, there will be an escape of gas, and if the opening in the lung has closed, the intrapleural pressure will become progressively less positive and then more negative. To obviate this, the Wolff's bottle must be replaced by a horizontal spiral of some four turns of narrow glass tubing, the distal end of which opens into the base of a broad cylindrical receiver. The spiral coils should be $1\frac{1}{2}$ inches in diameter. The receiver must be broad, so that when the fluid is displaced into it from the spiral tubing, the depth of water is not increased more than half an inch. Above it is in direct communication with the open air (Fig. 30). The spiral and receiver must be below the level of the patient. The intrapleural pressure is regulated by the extent to which these are filled with water. The water in the succession of loops of tubing prevents the escape of gas when the pressure in the pleural cavity is momentarily, even though considerably, raised by acts such as sneezing and coughing. When, however, there is a steady rise of pressure, the water is displaced from the spiral into the receiver and the gas escapes until the intrapleural pressure is again equal

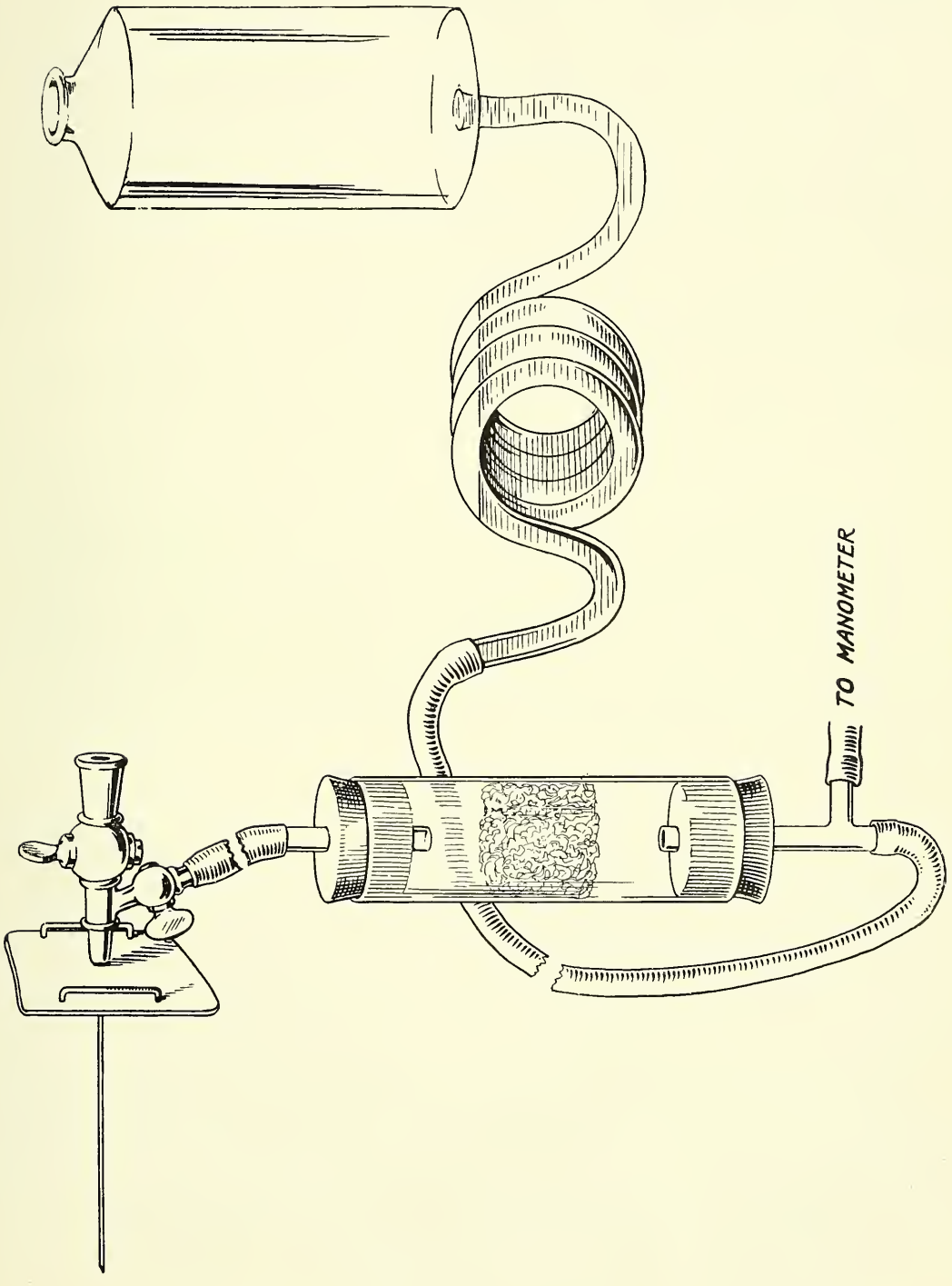

Fig. 30.

to the height of the column of water."* When such apparatus or that necessary for making an efficient substitute is not available, the following method of relieving the pressure must be used.

A large Record needle is driven through the chest wall. This needle can be maintained *in situ* by strands of sterilised thread tied round the head of it; the free ends of the thread are fixed to the chest by strapping some distance away. The outer end of the needle is covered with a few layers of gauze moistened in some antiseptic solution. By this means the intrapleural pressure will remain at about zero and the dangers of an ingravescent pneumothorax will be avoided. The immobility and compression of the lung will not, however, be complete, and the opening in it will take longer to close and may even fail to heal; this, however, is the lesser evil. Every twelve hours the needle should be removed, and should not be put back until signs of increasing intrapleural pressure indicate the need for it.†

The treatment of an opening in the surface of the lung which is kept patent by adhesions has already been described on p. 67.

Hæmothorax.

A collection of blood in the pleural cavity is almost always the result of injury to the chest wall or to the intrathoracic organs. The subject is dealt with fully in the chapter on Injuries and Gunshot Wounds.

A hæmorrhagic effusion is common in association with malignant disease of the pleura, but may occur also in cases of inflammatory or of tuberculous pleurisies, or as a complication of Bright's disease, cirrhosis of the liver and certain disorders of the blood. The treatment of a hæmorrhagic effusion differs in no way from that of a serous effusion.

Chylothorax.

This is a very rare condition; it may occur on either side of the chest or be bilateral. Chylothorax is due to the escape of chyle from the thoracic duct, as a result of trauma or disease such as involvement of the duct by primary or secondary malignant growth, pressure by enlarged glands, or thrombosis of the left subclavian vein.

The symptoms and signs are those of the primary condition combined with those of a pleural effusion. The character of the fluid cannot be diagnosed except after aspiration.

Treatment is symptomatic only and consists in the complete removal of the chyle, whenever it has accumulated sufficiently to cause symptoms. A negative pressure must not be produced in the pleural cavity, as this will

* *Quarterly Journal of Medicine*, April, 1918.

† The needle must not be replaced through the same wound and the new track must be anæsthetised on each occasion.

encourage the further escape of the chyle. For this reason the fluid is replaced by nitrogen (which is absorbed more slowly than oxygen), and the pressure in the pleural cavity at the end of the procedure should be equal to +5 mm. of mercury.*

In a case of chylothorax associated with a localised traumatic lesion, if spontaneous cure does not take place and the constant escape of chyle threatens the patient's life and necessitates repeated aspiration, the thoracic duct in the affected region should be explored, and the opening closed by sutures, if found. The route of access and the after treatment are similar to that described on pp. 235 and 236.

Tumours of the Pleura.

All tumours of the pleura are rare, and the benign forms even more so than the malignant. Three varieties must be considered—the benign, the mixed and the malignant.

The benign forms include lipoma, chondroma, fibroma and adenoma, and the mixed tumours chondro-myxo sarcoma and fibro-sarcoma. Whilst these latter increase steadily in size until they produce a fairly typical clinical picture, the growth of the former is seldom sufficient to give rise to any manifestations. The symptoms when present are due to the compression and displacement of the lung, and to pressure on the mediastinum causing engorgement and dilatation of the veins, cyanosis, dyspnœa and displacement of the heart. Owing to the absence of an associated pleural effusion, the diagnosis may be confirmed with the X-rays by the recognition of the lobulated outline of the tumour. An enlargement of the thyroid may be mistaken for a tumour of the pleura. This is likely to occur when the increase of the goitre is irregular and is mainly from the lower pole of one lobe of the thyroid. The tumour may then extend a considerable distance into the thorax, displacing the pleura and the lung in a downward and outward direction.†

The malignant tumours are divisible into two groups—those which originate primarily from the pleural membrane, and those which are secondary to a growth in some other part of the body. Secondary growths form smooth rounded masses scattered over the visceral and parietal pleura ; with increase in size, they coalesce. Effusions are not common except in the later stages. The symptoms, cough and pain, and later dyspnœa, are usually present before any physical signs can be recognised.

Primary malignant tumours are almost always endothelial in character, originating in the lining of the pleural membrane ; they may be sarcomatous. The former consist of a single or of multiple plaques of varying thickness ; the latter grow as a single lobulated mass. These tumours are almost

* For technique see aspiration with oxygen replacement, pp. 46 *et seq.*

† I am indebted to Mr. Wilfred Trotter for calling my attention to this condition, and for showing me a very striking case of this nature.

invariably complicated by an effusion which often at first is pale yellow, but becomes hæmorrhagic in the later stages. The symptoms are pain, cough and dyspnœa; the last may be present even when there is no effusion. The fingers are, as a rule, clubbed. There is dilatation of the veins and œdema of the overlying tissues when the chest wall is invaded. The signs are usually those of fluid, and diagnosis is rarely possible until the effusion has been completely removed by oxygen replacement. A persistence then of increased resistance, of dullness and of diminished or absent breath sounds over a localised area of the chest wall, is suggestive of the nature of the lesion, whilst the shadow given by the X-rays may confirm the diagnosis. The malignant or benign character of the tumour can be surmised only; the former variety is more common, is more rapid in its progress and is more frequently complicated by fluid.

TREATMENT.

The removal of benign tumours, unless very large, is always possible; of mixed tumours is occasionally practicable, and when accomplished is followed by good results; of sarcomata, is feasible, but only when the growth is small and localised to the pleural membrane; endotheliomata are inoperable by the time the diagnosis can be made.

The fluid, when present, must be completely removed by oxygen replacement and the situation of the tumour accurately localised. The patient is anæsthetised with ether by the intratracheal method. An incision is made through the intercostal space immediately below (or above) the tumour and the mass explored with the finger so as to determine the exact extent and mobility of it. A small portion of the growth is excised for immediate macroscopic and microscopic examination. If the tumour is benign, or is a chondro- or myxo-sarcoma, the operation is continued. A flap including all the soft tissues superficial to the ribs is turned back off that part of the chest which overlies the growth. The flap should be at least half an inch bigger in all directions than the tumour and the upper and lower borders should run parallel with the ribs. The peripheral ends of the ribs are divided and the intercostal muscles including the vessels and nerves are cut through at the level of the edges of the skin incision. The proximal ends of the exposed ribs are cracked through with bone forceps and the second flap consisting of ribs and intercostal muscles is stripped off the pleura and turned back. The original opening into the pleura is enlarged and the whole of the tumour is removed by cutting through the pleura at a distance half an inch from the growth. The osteo-muscular and cutaneous flaps are returned and stitched into place, care being taken to get accurate re-apposition of the ribs. The air in the pleural cavity is then replaced by oxygen at a negative pressure equal to about 10 mm. of mercury. An effusion may develop during the next few days. If it is large enough to cause symptoms or is under pressure, it must be treated by oxygen replacement.

CHAPTER V.

INJURIES OF THE LUNGS AND PLEURA, INCLUDING GUNSHOT WOUNDS.

HERNIA OF THE LUNG. DIAPHRAGMATIC HERNIA.

There are certain rare complications which may supervene immediately on an injury of the lung or pleura irrespective of the severity of it. In most of the cases of this nature which have been recorded, the trauma was comparatively trivial. The symptoms are characteristic either—

(1) Of an involvement of the cardio-vascular system—syncope, the result probably of a reflex from the parietal pleura.

(2) Of a cerebral lesion—involvement of consciousness, convulsions and paresis ; or

(3) Of a local nerve injury—paralysis of one or more groups of muscles.

Since these phenomena are of a serious character and may appear as a direct result of surgical treatment of the simplest nature, or as a complication of graver injuries, there is a special interest attached to them.

Pleural Reflex and Gas Embolism.

These two apparently widely different conditions are discussed together because certain definite groups of symptoms following on injuries of the pleura or of the lung are considered by some authors to be caused by gas embolism but by others are attributed to a pleural reflex.

There are three types of clinical phenomena, all of which have one common characteristic, namely, immediate onset after the injury :—

(1) Paresis or paralysis, sudden in onset and affecting one or more groups of muscles, the whole of a limb or half the body. This

gradually disappears after weeks or months. There may or may not be loss of consciousness at the onset.

(2) Convulsions, clonic or tonic, general or localised, associated with loss of consciousness. This may be followed by paresis or paralysis.

(3) Syncope, which may result in almost instantaneous death, or, after a variable period, subside and be followed by complete recovery.

The first two types are extremely rare and occur usually after injuries in which there is damage to the lung as well as to the pleura ; the injury being of the nature of a crush or bullet wound. Orlowski and Fofanow,* however, describe a case of pleural "eclampsia" in which, a few minutes after the production of an artificial pneumothorax, there was loss of consciousness, vomiting, spasm in certain groups of muscles, arrest of respiration and extremely weak action of the heart. These symptoms subsided after a short time, but recurred frequently in the course of the next twenty-four hours. The acuter manifestations then ceased and the patient was left with a paresis of the affected muscles which recovered gradually in the course of a few months. The pathological findings have been so few and so indefinite that the supposition that the manifestations are caused by gas embolism is based mainly on the clinical picture. So far as the paralytic phenomena unassociated with loss of consciousness or clonic spasms of the muscles are concerned, specially when they are monoplegic or hemiplegic in character, it is probable that some of the cases are functional in character, or due to shock to the brachial plexus. This is particularly the case when the paralysis is of the arm on the same side as the lesion of the chest. Von Saar,† in his experiments, has occasionally obtained movements of the upper limb by stimulation of the parietal pleura. This result could not be obtained after cocainisation of the intercostal nerves ; he regards it therefore as a reflex phenomenon.

Syncope is a relatively much commoner symptom and may occur as the result either of puncture of the pleura or of irrigation of the pleural cavity. The most striking feature about it is the apparently trivial nature of the disturbance on which it usually ensues. It is extremely difficult to ascertain what combination of circumstances is necessary for the production of it. It is more often seen during puncture of a normal than of an inflamed and thickened pleura. In most of the recorded cases it has occurred at the moment of the introduction of the needle for the injection of gas in the treatment of phthisis, and in many of these cases the parietal pleura was

* "Tuberkules." Jg. 2, Hg., s. 386.

† "Uber pleurogene Extremitätenreflexe." Verhand. d. deut. Gesell. f. Chir. 1912, II., s. 361.

thin and free of adhesions, and previous injections of gas had been made (in one case fifteen).* Syncope is occasionally produced, however, by the injury to the parietal pleura caused by the passage of the needle when exploring or aspirating a simple or pyogenic effusion. It probably never occurs when, before the puncture, the track of the needle is efficiently anæsthetised. Syncope has been observed during irrigation of the pleural cavity. In many cases it has been associated with a sudden rise in intrapleural pressure, but it has occurred even when every precaution has been taken to ensure no alteration of tension. The manifestations produced by irrigation are less severe than those which follow on trauma of the membrane; there is intense giddiness and nausea, rarely vomiting or actual loss of consciousness.

Riedinger† and Sauerbruch‡ both describe a clinical picture which has been observed to follow a blow on the chest and is called by Sauerbruch "commotio thoracis." It is compared by them with concussion of the brain. The patient gives a loud inspiration and falls apparently lifeless to the ground. The force of the heart beat is minimal, the face pale and cold, there is often vomiting and sweating. This condition of shock may end in death. Riedinger, as the result of experimental work, states that the chief symptoms are due to fall of blood pressure caused by direct stimulation of the intrathoracic vagus and maintained by paralysis of the sympathetic. "Commotio thoracis" cannot be regarded as a name happily chosen and the implied correspondence with concussion of the brain seems to involve views of the pathology of the latter condition scarcely in accord with modern opinion.

To summarise: Trauma of the parietal pleura and of the lung is very occasionally followed by certain general manifestations of abrupt onset. The least uncommon is the syncopal attack, which is probably a reflex, set going by stimulation of the extremely sensitive parietal pleura. Following laceration of the lung, air may enter the veins and cause a gas embolism of the brain or spinal cord: the symptoms of this may be disturbance of consciousness, tonic and clonic spasms of groups of muscles followed by paresis or paralysis. Some of the sequelæ of trauma may be ascribed to concussion or injury of the brachial plexus; others are undoubtedly functional.

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* The only case of pleural syncope which I have seen occurred at the moment of puncturing a perfectly healthy parietal pleura. The track of the needle had been imperfectly anæsthetised and as the needle penetrated the pleura, the patient gave a slight start and cry from the pain; there was immediate loss of consciousness, marked pallor, and the pulse and respiration became imperceptible. This condition lasted some 30 seconds, when the pulse, colour, and consciousness returned. There were no after effects and numerous subsequent punctures of the pleura, which were painless, were unattended with any abnormal manifestations.

† "Uber Brusterschutterung." Festschrift der Alma Julia Maximiliana, Wurzburg. 1882, s. 221.

‡ "Die Bedeutung des Mediastinal emphysems in der Pathologie des Spannungs-pneumothorax." *Bruns Beitrage*, Bd. 60, 1908, s. 450.

INTRATHORACIC INJURIES.

Injuries of the lung are as a rule arranged and classified according to the method of production or the nature of the instrument responsible for the trauma. This is unsatisfactory, as the same type of lesion may be occasioned by any one of several means. It is of much more importance to understand the various changes in the physiological and mechanical conditions and the morbid and pathological processes which may be produced, since a know edge of these must form the basis for the treatment—not of the injury itself—but of the abnormal states which are the immediate or later results of such injury. We have then to consider :—

A. The changes and effects produced by—
   1. Compression of the chest.
   2. Contusion of the pleura or lung. With these must be considered the complications due to infection and those due to fractures of the bony framework.
   3. Laceration of the lung.

B. Laceration of the chest wall causing communication between the outer air and the thoracic contents.

C. The complications arising from injuries to adjacent organs and structures.*

A. 1. Compression of the Chest.

The effect of compression of the chest is in one respect similar in all people—it produces an increase in the intrapleural pressure, which increase may be very sudden and very considerable. If the compression is unilateral, it will cause considerable dislocation of the mediastinum and its contents. Owing to the resiliency of the chest wall in young people, there is less likelihood of fracture of the skeletal framework and on the cessation of the compressing force the chest will recover its shape, and the intrathoracic tension will return, for a period at any rate, to the normal. In adults, fracture of the ribs and sternum and the complications due to such lesions are more common.

Traumatic asphyxia (masque ecchymotique) is the name given to the symptom-complex resulting from sudden very great but temporary compression of the chest, such as may be occasioned in panics or in buffer accidents. The symptoms are caused not by the blow but by the intense and sudden rise of the intrapleural pressure in both sides of the chest, and by the effect of this on the venous circulation. The return of the blood to the heart is to a great extent dependent on the negative pressure in the chest. As a result of the abrupt change in pressure, the flow

* It is not proposed to deal in this book with the cases which come under group C.

of the venous circulation is not only checked but the blood in the intra-thoracic veins at the moment of the compression is actually driven backwards, while the supply from the arterial capillaries is undiminished. The veins of the neck and head (with the exception of the intracranial and intraoccular ones)* and, to a less extent, of the upper extremities are therefore suddenly and intensely engorged. The venous capillaries are unable to stand the pressure and rupture. The space available in the veins below the chest is practically unlimited, therefore no effects are produced on the abdomen and lower limbs. The space available above the site of the compression is, on the other hand, distinctly limited (none at all is available within the skull), consequently all the effects of the compression are manifested in this region.

The clinical manifestations are absolutely characteristic. Immediately after the accident, the upper part of the body is intensely congested and is purple in colour as the result of the extravasation of blood. The tissues of the face and neck are the most implicated,† subconjunctival and, to a less extent, submucous hæmorrhages are present. The extent of the suffusion of blood is controlled by the degree of support to the outer side of the walls of the capillaries afforded by the clothes ; so that a hat or cap worn at the time of the accident is sufficient to prevent any extravasation over the top of the head, and round the forehead will be a sharp line of demarcation between the purple skin below the brim of the hat and the normal skin above. A similar effect is produced by the pressure of the collar, and even by the rim of a pair of spectacles. The influence of external support in preventing rupture of the capillaries accounts also for the absence of greater extravasation of blood into the submucous tissues of the mouth. As the colour is due to extravasation of blood and not to a hyperæmia, pressure on the skin produces no change. There is usually considerable œdema of the eyelids and of the conjunctiva. Transitory blindness may occur, and, rarely, a permanent impairment of vision owing to optic atrophy.

There may be, at the moment of the accident, a temporary loss of consciousness due to the sudden momentary intracranial venous stasis. Cerebral and meningeal hæmorrhages have never been found at autopsy. The nature of the injury is in itself sufficient to disturb for the time being the normal rhythm of respiration and the action of the heart. In addition, there is the influence of shock and later the effects of pulmonary congestion.

Traumatic asphyxia is usually seen in young persons only ; in elderly people the force of the compression would probably cause fracture of the sternum and other serious injuries liable to be fatal in themselves. If the

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*A rise of venous blood pressure inside the skull is necessarily accompanied by a corresponding rise of intracraneal pressure which supports the outside of the wall of the venous channel.

† The implication of the upper limbs is much less extensive, owing to the greater efficiency of the valves in the veins.

shock is survived and there are no serious complications, the condition of traumatic asphyxia is not usually fatal and in one or two weeks after the injury the patient will have recovered his normal appearance.

Rupture of the bronchus. Rupture of one of the primary bronchi is probably due to the compressing force on the chest (and consequently the rise of intrapleural pressure) being at the moment of onset unilateral. This would occasion a violent dislocation of the mediastinum to the opposite side and a rupture of the bronchus on the same side as the lesion. The laceration in the bronchus is extrapleural and the more immediate

Fig. 31. Traumatic pneumothorax. The lung was healthy and collapsed therefore as a ball round the hilum. Re-expansion is taking place. (*Cf.* Fig. 25.)

effect (when death from shock or from associated injury to some vital organ is not instantaneous) is an intense mediastinal emphysema which is in most cases fatal. If there is a laceration of the pleura at the line of reflection from the lung to the mediastinum and chest wall, then the mediastinal emphysema is complicated by a pneumothorax or even a hæmo-pneumothorax.

Laceration of the lung does not usually occur as a result of the rise in intrapleural pressure unless there is a simultaneous rupture of some part of the containing wall, such as fracture of a rib or rupture of the diaphragm. A laceration may, however, occur without fracture when localised adhesions interfere with the dislocating action on the lung of the compressing force.

A compressing force applied to the front and back of the chest causes increase in the curve of the ribs, which break somewhere in the region of the axillary line, the fractured ends projecting outwards away from the pleura. Lateral compression of the chest or direct blows usually drive the fractured ribs inwards and cause direct injury to the soft tissues.

The occurrence of contusion or laceration of the lung necessarily implies some degree of disorganisation of the normal defences of the body against the inroad of the infective organisms. There are many injuries which are capable of producing these changes in which, however, the compressing force is so small or so localised as to be negligible, and for this reason these morbid and pathological processes are dealt with under independent headings.

2. Contusions of the Pleura and Lung.

Contusions of the pleura give rise to a local dry pleurisy, or pleurisy with effusion : the pleurisy is likely to be considerably aggravated if the contusion is complicated by fracture of a rib. These conditions have been fully dealt with in the previous chapter.

A contusion of the lung causes a local extravasation of blood into the tissues and alveoli : the irritation due to the blood is followed by a mild inflammatory reaction of the surrounding tissues and the production therefore of an area of pneumonic consolidation. The symptoms are slight rise of temperature, with cough and expectoration. Streaks of blood may appear in the sputum immediately after the injury as a result of the rupture of vessels ; after two or three days the sputum acquires the rusty appearance common in cases of pneumonia. The primary effects of contusion are rarely serious, but the damage which has been done to the parenchyma and to the lining of the alveoli and of the bronchioles and the interference with the blood supply frequently allows the penetration into these injured parts and the proliferation of the various organisms which are always to be found inhabiting the upper air passages. The character of the infection which may develop and the extent to which it may spread varies very considerably. The less severe infections manifest themselves as a localised, probably a lobar, pneumonia : in some cases the spread extends throughout the lung and there is a complete unilateral pneumonia. The more acute infections may result in abscess formation or gangrene.

Massive collapse. This condition is not frequently seen in civil life except as a post operative complication ; but it is possible that the fewness of the recorded cases is due to absence of a general recognition of the morbid processes and of the clinical phenomena which are associated therewith. Numerous cases, however, have been recorded as occurring after gunshot wounds. Massive collapse involves a lobe or the entire lung and is

characterised by a very great reduction in the size of the organ. This extensive shrinkage is due on the one hand to the contraction of the elastic fibres, and on the other hand to the active absorption by the blood-vessels of the air from the alveoli.

The factor which determines massive collapse is uncertain. There are three main hypotheses : (1) That the collapse is due to the inhibition of the respiratory muscles. (2) That the primary cause is the obstruction of the bronchi or bronchioles by a plug or a series of plugs of mucus. (3) That the responsible factor is an acute reflex spasm of the muscles of the bronchioles. There is very little evidence in favour of the first of these explanations. As regards the second, it is necessary to suppose the complete obstruction of a large bronchus or the simultaneous obstruction of numerous bronchioles and at the same time that secretion should be present and of the right consistency. Obstruction of a bronchiole does occur in certain infective conditions. It is seen in cases of chronic phthisis and of bronchiectasis with a chronic abscess cavity, but it is probable that the plug of mucus is of considerably less account in determining the occlusion of the bronchial tube than is œdema. Now the mucous lining of the bronchioles consists of tissues which may easily become œdematous and a comparatively little œdema will produce a very considerable constriction and even obliteration of the lumen. Œdema, moreover, is the change we would expect to find as a result of the irritation of trauma, the irritation of anæsthetics, or the irritation of infection. It is more than probable that each or any of the other suggested causes may be a subsidiary factor : paralysis or paresis of the inspiratory muscles abolishes or diminishes the power of suction of the air through the constricted bronchioles, a very little mucus will quite easily suffice to obliterate the passage of air through the approximated walls ; a spasm of the bronchioles would have the same effect.

From our knowledge that in massive collapse the main primary change is an intense and rapid diminution in volume of the lung, it is possible to deduce what are the secondary intrathoracic changes and what must be the clinical manifestations. The rapid contraction of the lung implies the rapid production of a high negative pressure. The falling in of the chest wall and the rising of the diaphragm compensate for this to but a small extent ; the mediastinum and heart are therefore violently drawn over to the affected side and the opposite lung becomes distended and emphysematous.

The intensity of the symptoms is in proportion to the rapidity of the occurrence and to the extensiveness of the massive collapse. There is pain, great dyspnœa and cyanosis, the pulse is increased in rate and may be irregular, in severe cases the patient is collapsed ; there is diminished resonance, dullness and weak breath sounds, and later tubular breathing. Within twenty-four to forty-eight hours the symptoms usually subside and after four or five days there is very little, if any, evidence of the intense intrathoracic disturbance.

3. Lacerations of the Lung and Pleura.

Rupture of the lung may arise either with or without fracture of the bony framework of the chest. The latter occurs more commonly in children in whom the elasticity of the ribs and cartilages is greater. Rupture of the lung nearly always involves the visceral pleura and there is therefore an escape of air or blood, usually of both, into the pleural cavity. If the tear communicates with one of the bronchioles, there will be hæmoptysis; if with the tissues of the chest or mediastinum, there will be emphysema. The clinical manifestations which these changes give rise to are obvious and characteristic, but there are in addition certain morbid changes which take place in the lung round the seat of the injury—hæmorrhagic infiltration, consolidation, reaction of the tissues, lowered vitality and loss of power of resistance against infection. These give but few distinctive symptoms, while the signs are obscured by the intervening hæmo- or hæmo-pneumothorax.

Hæmoptysis. It has already been stated that hæmoptysis does not necessarily imply laceration of the lung, nor is it a constant symptom of that lesion. This statement is equally true whether the injury is caused by a blow, a crush, or a gunshot wound. Hæmoptysis is rarely fatal unless the rupture is at the hilum and there is direct communication between a bronchus and a large vessel, or unless there is extensive laceration of a lung which is unable to collapse owing to the presence of adhesions.

Hæmothorax. The size of the hæmothorax depends on the freedom of the pleura from adhesions, on the extent of the laceration of the lung tissue and on the nearness of the lesion to the hilum. The arrest of the bleeding is due to the collapse of the lung (in the absence of adhesions), or to the compression of it by the escaping blood and air (when the membranes are in part adherent), and possibly also to the deposit of fibrin on the pleural surfaces. Henry and Elliott,* describing the aseptic hæmothorax due to gunshot wounds, state that "as the blood flows from the wound into the pleural cavity it clots rapidly. But the clotting is not a massive coagulation . . . fibrin is thrown out and becomes deposited in layers of varying thickness both on the parietal pleura and on that part of the lung which dips into the effused blood. This fibrin may be deposited in one continuous layer or in patches, and it is to be found in great amount, and often with a more massive coagulum resembling ordinary blood clot, in the diaphragmatic pleural recess and posteriorly in the concavity of the ribs. At first the layer can be readily stripped off the serous surfaces, but later it becomes organised and firmly adherent to the pleura.

"The deposition and organisation of the precipitated fibrin are at first of considerable advantage to the individual, for the process may not only

* "The Morbid Anatomy of Wounds of the Thorax," based on the collection of the records of 100 fatal cases, by Captain Henry and Major Elliott.

seal up the wound in the lung, but it may also prevent the subsequent spread of sepsis from a damaged and infected respiratory tract into the pleural cavity.

"The throwing out of the fibrin leaves in the thoracic cavity a fluid which consists of blood serum together with practically all the usual cellular elements of the blood, but which has at this stage no power of clotting because it contains no fibrinogen . . . later, however, after a period of time which varies greatly in individual cases, an inflammatory pleural exudate is thrown out and added to the pool of liquid blood. If much fibrin ferment is still present in the hæmothorax after the primary clotting, the fibrinogen of this exudate will also be coagulated within the thorax and so increase the density of the fibrin layer enveloping the lungs."

The symptoms and signs of a hæmothorax resemble closely those of a pleural effusion, but certain differences exist which require special notice. Dyspnœa is almost always present and is intense. In all cases where there is a considerable accumulation and absorption of blood, there is some fever, variable in degree but rarely causing a rise in temperature above 102° F. If the case is seen soon after the injury, the dullness and resistance over the affected side of the chest are not so marked as one would expect in comparison with the displacement of the heart and the dyspnœa. This is due, in part, to the blood being frothed by the air which escapes at the same time and containing, therefore, innumerable small bubbles of gas. This admixture of air with the blood is also responsible for the early marked displacement of the heart and for its subsequent partial return in the next two or three days as the gas is absorbed. A late increase in the amount of fluid is usually due to a secondary effusion, which may be simple or infected.

In a large majority of cases of hæmothorax which are the result of gunshot wounds, the diaphragm on the affected side is found to be raised instead of depressed, as one would expect, by the weight of the fluid. There are two possible explanations. One is that the phrenic nerve, which is comparatively unsupported in its passage through the thorax, is particularly susceptible to the concussion effects of the missile and that the elevation of the diaphragm is the result of the paralysis. The second suggested explanation is that the collapse of the lung following on the impact of the foreign body causes at first a considerable negative intrapleural pressure. The evidence that this is the case is small and even if such negative pressure were established it would be rapidly changed by the increasing hæmo- or hæmo-pneumothorax.

Pneumo- and hæmo-pneumothorax. Air alone without blood in the pleural cavity is only a rare consequence of injury. In most cases of laceration of the lung there is, at the onset, some escape of gas; but this is rapidly absorbed. Occasionally there is a large and persistent hæmo-pneumothorax or even pneumothorax which may be of the ingravescent type.

Simple pneumothorax as a result of gunshot wounds is comparatively rare. The majority of cases which have been observed have developed several days after the injury. They are due probably to the escape of gas through a part of the lung surface which was weakened, but not actually destroyed, at the time of the injury.

Infection of the hæmothorax. The blood in the pleural cavity may become infected through the wound in the lung or by a foreign body penetrating from without. Reference will be made later to the dangers of

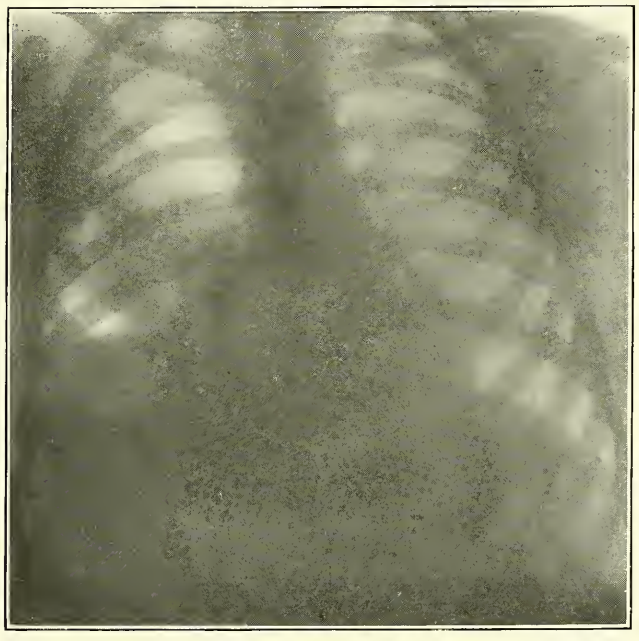

Fig. 32. Traumatic hæmo-pneumothorax due to injury of the chest wall with fracture of the rib. The healthy lung has collapsed as a ball round the hilum. (*Cf.* Fig. 31.)

infection of the lung parenchyma. A foreign body (*i.e.*, bullet or metal fragment) penetrating the pleural cavity or possibly the lung carries with it particles of clothing or other infective material. This may become lodged on the surface of the pleura and the blood tends at once to enclose the septic focus by the formation of a voluminous clot around it. The organisms, usually anærobic ones or streptococci, flourish in the centre of this clot, and gradually pervading it, finally reach the surface and produce a general infection of the pleural cavity. In these cases the clinical picture is peculiar, but once recognised is quite characteristic. There is at first a toxæmia

as the result of slow absorption from the infected nucleus of the blood clot. But the hæmothorax appears to be a simple one and aspiration withdraws a fluid which is sterile. The symptoms of septic absorption increase and the patient is more ill than can be accounted for by the pathological and mechanical changes which accompany an aseptic hæmothorax. Quite suddenly, and this coincides with the spread of the organisms through the surface of the clot, there is an exacerbation of the symptoms and aspiration will now reveal a pyothorax. If the exacerbation of the symptoms is associated with a further rapid displacement of the heart, the presence in the pleural cavity of anærobic gas-producing organisms must be suspected. In some cases the infection is acute, and disseminates from the beginning.

When the infection reaches the hæmothorax from a primary focus in the lung, the clinical phenomena due to the invasion of the hæmothorax by the organisms are less easily recognised, as the patient is already suffering from toxic absorption from the lung.

Lacerations of the pleura. The manifestations which are the direct outcome of lacerations of the pleura are similar to those resulting from contusions of the membrane, though usually more severe in character. They are generally obscured by the graver symptoms due to associated laceration of the lung. There are, however, three possible complications of this lesion : (1) The gas from a pneumothorax, or directly from an adherent lung, may escape into the cellular tissues and give rise to surgical emphysema ; (2) Blood from an injured intercostal or internal mammary vessel may escape into the pleural cavity. (3) The infection can more easily spread into the pleural cavity from a septic focus in the chest wall, or into the cellular tissues from a pyothorax.

In uncomplicated cases, the lung will almost certainly become adherent to the chest wall at the seat of the laceration.

Surgical emphysema is due to the escape of air into the tissues either of the chest wall or of the mediastinum. In the former case, the air enters through a rupture in the parietal pleura, either from a pneumothorax or directly from an opening in an adherent lung. In the latter case there is communication from a bronchus, or from a pneumothorax through a rupture in the pleura. Emphysema of the cellular tissues of the chest wall may be localised to the region of the wound or may spread thence along the subcutaneous tissues and even among the intermuscular planes, involving in severe cases the whole of the face and the limbs down to the wrists and ankles. A sensation of fine crepitation is felt when the skin is pressed on, and a resonant note is obtained on percussion ; the condition is, as such, in no way dangerous. When, however, the distension of the skin is very great, sloughing may occur unless the tension is reduced by incisions. The gas is absorbed in a few days.

Mediastinal emphysema is much more dangerous, especially when the gas escapes directly into the tissues from a bronchus. The distension may be such that there is compression of all the structures of the mediastinum, and especially of the veins. There is progressive dyspnœa, cyanosis and distension of the veins of the face and neck, and failure of the heart. The condition is recognised by the symptoms just mentioned, by the tympanitic note on percussing the sternum, and later, by the spread of the emphysema into the root of the neck.

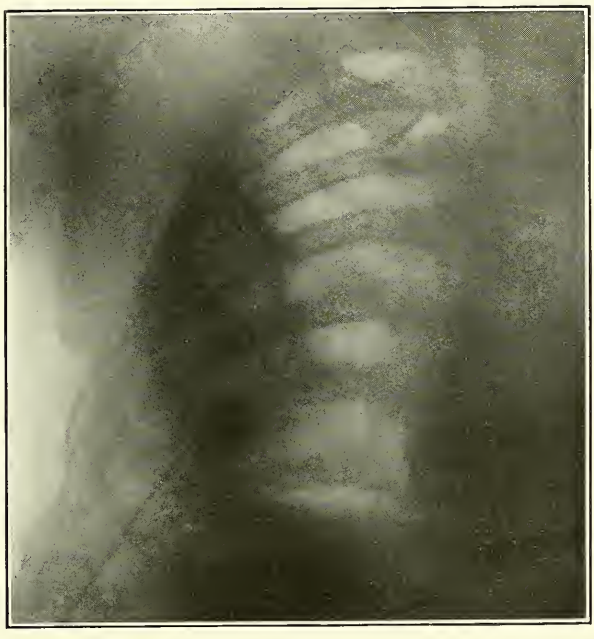

Fig. 33. Chronic discharging empyema due to shell fragment in the pleural cavity. The probe shows the position of the sinus.

Changes in the Lung due to Penetration by Foreign Bodies.

Describing the morbid changes found in the lungs as a result of gunshot wounds Duval* states that the whole length of the wound in the lung shows a more or less widespread mortification of the cellular and parenchymatous tissues. The lung substance is pneumonic and infiltrated with blood. The hæmorrhagic infiltration is in the nature of a traumatic infarct; it may be limited to the track or involve the greater part or the whole of the lobe. In the track may be the metallic fragment or other foreign bodies such as pieces of the ribs or scapula. These bony fragments may be embedded

* "Les Plaies de Guerre du Poumon," par Pierre Duval, 1917.

in the lung to a depth of 5 cm.. They are particularly dangerous, either immediately by producing laceration of vessels or later owing to infection and secondary hæmorrhage.

Morbid changes at a distance from the track are not uncommon, the lower lobe may be airless as a result of compression by a hæmothorax, whilst the apex of the lung shows compensatory emphysema. Hæmorrhagic infiltration due to *contre coup* may be found in the lung of the same or of the opposite side also.

The track of the metal fragment or bullet is practically always infected, either immediately as a result of contamination by the foreign body or later by the invasion of organisms from the upper air passages. The seriousness of this condition varies greatly. A generalised pneumonia or a localised gangrene or abscess is not uncommon and increases the danger of secondary hæmorrhage. But of far greater seriousness is the risk of the spread of infection from the track outwards to the surface of the pleura and the resultant septic processes in that cavity.

Excluding the mortality during the first twelve hours, by far the greatest number of deaths following gunshot wounds of the chest are due to sepsis. There are two periods at which this complication develops. The first is on the second or third day and it is then the result of direct infection of the lung or of the pleural cavity. The second period is about the seventh day and is due to the invasion of the hæmothorax by the spread of organisms from the track in the lung.

It is doubtful if gas gangrene ever occurs, the richness of the blood supply being an efficient prophylactic to the development of the anærobes. Gas gangrene of the pleura is rare.

Sphacelation is an unusual occurrence ; it is due to the combination of laceration of the lung and of sepsis. When part of the lung has been extensively torn as a result either of a crush or of a gunshot wound, that partially separated portion may, in the presence of infection, become gangrenous and slough off from the main part of the organ. This is the only form in which anything resembling a gangrene of primary vascular origin occurs in the lung.

Thus far discussion has been devoted to the abnormal changes which are found as the result of various injuries of the lungs and pleura and of the phenomena which these give rise to. Certain manifestations liable to occur in all injuries are sufficiently frequent and important to deserve some special consideration from the more definitely clinical aspect. Such conditions are shock, fever and dyspnœa.

The severity of the *shock* depends on the extent of the lesion and on the amount of injury which has been done to sensitive areas such as the pleura. A clean bullet wound through the lung may have practically no influence on the blood pressure ; a severe injury to the pleura is followed by

considerable shock, and if to this is added laceration of the lung and hæmorrhage, the degree of shock is still greater. Children are apt to show a disproportionately severe degree of shock in cases of injury to the chest. Sometimes in them shock of alarming gravity develops when the structural damage discoverable is limited to bruising.

Fever, in cases of injury to the lung, is not necessarily an indication of suppuration. The temperature associated with suppuration is usually characterised by considerable diurnal variations. Fever which is maintained is generally seen in cases of less severe infections such as cause pneumonic consolidation or pleurisy. But even when there is no infection and in the early stage after an injury which has caused contamination of the tissues and before the organisms have had time to develop, a rise of temperature is an almost constant phenomenon. This febrile state may, however, be masked by the degree of shock which the patient has sustained. A rise of temperature is frequently the result of absorption of the products of septic decomposition, but it may also follow absorption of disintegrated tissue (*e.g.*, blood) when such tissue is absolutely sterile, and it may be produced as a consequence of the reaction of the tissues to irritation. The frequency of fever in cases of injury of the lung is probably accounted for by the richness of the blood supply.

Dyspnœa is one of the most striking symptoms of injuries of the chest. It may be said that an injury unaccompanied by dyspnœa is very unlikely to be serious. It is usually of immediate onset and occurs almost equally in cases of wounds of the chest wall only and wounds implicating the lung as well. The shortness of breath is most intense for the first hour or two after the injury, and gradually diminishes during the ensuing twenty-four to forty-eight hours ; this diminution is noticeable even in those cases in which there is a slow progressive displacement of the lung by a hydro- or hæmo-thorax. Later the dyspnœa may again increase. This is specially so when an ingravescent pneumothorax occurs or there is extensive or progressive pneumonic infiltration. There are four possible explanations of the primary dyspnœa : (1) That it is due to the cessation of movements on the affected side of the chest by a reflex protective mechanism ; (2) that there is paralysis of movement from the shock to the nerves ; (3) that it is the result of rapid formation of a large hæmothorax or hæmo-pneumothorax with associated collapse of the lung on the same side and possible compression of the opposite lung also—the later diminution of the breathlessness can be accounted for by the rapid absorption of the gas, particularly the oxygen ; (4) that it is at times functional in character. The dyspnœa which is due to the rapid accumulation of blood or of blood and air in the pleural cavity can be immediately relieved by aspiration with oxygen replacement, and the dyspnœa which is of a functional character by morphia. It is therefore very necessary that the causes of this distressing symptom should be borne in mind so that, when possible, it may be relieved without delay.

B. Lacerations of the Chest Wall causing communication between the outer air and the thoracic contents.

A laceration which causes an opening in the chest wall introduces an entirely new physiological-mechanical factor and at the same time greatly increases the danger of sepsis, since infection in such cases is inevitable.

The physiological-mechanical factor has already been discussed in Chapter II. Briefly recapitulated, it consists in the fact that the air on the affected side of the chest is at atmospheric pressure, while there is a varying negative pressure in the opposite half of the thorax. The lung is collapsed, the mediastinum is displaced to the opposite side and flaps to and fro with each respiratory movement. If the opening is large and remains untreated, there is a grave risk of the patient dying of shock or of asphyxia.

TREATMENT.

There are three cardinal considerations in the treatment of all injuries of the chest. They are the counteraction of the shock, the arrest of the bleeding and the prevention of septic infection. A hypodermic injection of morphia should be one of the first things given, as it will abolish or reduce the pain and by this means prevent the aggravation of the shock, while at the same time it helps to check the bleeding. The shock should be treated on the usual lines, but it is inadvisable to give stimulants if they can be avoided, as the unnecessary raising of the blood pressure will encourage the bleeding.

If the patient is kept in a state of absolute rest it is only rarely that active intervention, apart from the morphia, is necessary for checking the hæmorrhage. The bleeding from a wound in the region of the hilum causing laceration of the larger vessels is, as a rule, uncontrollable and fatal. Laceration of the lung will cause either a hæmoptysis or hæmothorax or both. The pressure of the blood in the pleural cavity on the lung and the associated collapse of that organ is usually sufficient to stop further bleeding. This action is greatly assisted by the effect of the morphia on the blood pressure and its beneficial influence on the restlessness of the patient and on the dyspnœa which in these cases is often aggravated by the mental state. If, however, the main cause of the hæmorrhage is a wound of a vessel in the systemic circulation, ligature of that vessel may be the only means of controlling the loss of blood. It is often difficult to ascertain the source of the hæmorrhage, but if there is a wound of the chest wall in the neighbourhood of an artery and there is a constant escape of blood from that wound, or a hæmorrhagic infiltration of the tissues around it, then that artery must be suspect. It is occasionally extremely difficult to decide whether the intrapleural bleeding has been arrested or is continuing. In the former case, delay in active intervention is justifiable, and even advisable, in order that the patient may recover from a state of shock. If, on the

other hand, the hæmorrhage is progressive, too long a delay may result in the death of the patient. The blood pressure is the only certain means of distinguishing between collapse due to loss of blood and the similar state due to shock. A reading should be taken every half hour and if the blood pressure is found to be steadily dropping, it may be taken as certain that the bleeding is continuing and that an operation is urgently necessary.

The extent and the persistence of the bleeding is much more easily recognisable when there is communication through the chest wall from the pleural cavity. This communication may be a narrow one, allowing for the escape of blood outwards (and not the entrance of air), by reason of an intrapleural positive pressure. Or the opening may be large and there is a mixture of blood and air in the chest, the blood overflowing when it reaches the level of the opening. The danger to the patient from hæmorrhage depends on the existence of adhesions between the pleural surfaces. If the lung is free to collapse, bleeding will probably stop, but if part of the lung is fixed by adhesions, the wound is kept open and the loss of blood can be checked by operative means only.

It is of the utmost importance to remember that quietness and absence of movement are essential to success and that any attempt to move the patient during the first two days may have fatal consequences.

Infection is by far the most serious and the most fatal complication of injuries. It is obviously of the utmost importance, therefore, that every precaution should be taken against the infection of a wound which is not already contaminated, to limit and control the sepsis which has already developed and to guard against the possibility of secondary infection. The limitation of septic processes is possible only by early diagnosis and immediate treatment of the focus of suppuration. It must be borne in mind, moreover, that organisms may reach the injured tissues by conveyance on the instrument which has inflicted the wound, from an infected track in the chest wall, from an infected focus in the lung or from the upper air passages. War surgery has taught us the great advantages which are to be gained by excising contaminated and even infected wounds, but it is as yet scarcely appreciated to what extent infections of the lung may depend on the presence of organisms in the mouth and fauces, and that this may be diminished by the immediate and constant cleansing of these parts.

Treatment of Fractures.

A fractured rib unites with the greatest ease and the displacement of the fragments is rarely sufficiently serious as to necessitate energetic measures. Treatment is therefore practically confined to reducing the pain caused by the irritation of the broken ends to the soft tissues, especially to the parietal pleura. This is accomplished by immobilising and by strapping the affected side of the chest. A series of strips of plaster each slightly overlapping the

one above are firmly applied over the ribs. The upper and lower strips should be well above and below the injured ribs while the ends extend at least two inches beyond the middle line on the sound side. Occasionally this treatment increases instead of diminishes the pain and must then be discontinued. This is frequently the case when the fracture is of one of the lower free ribs.

When several adjacent ribs are broken, the deformity and the overlapping of the fractured ends may be considerable. In such cases it is advisable to expose the ribs and to fix the fragments by driving a steel pin (*e.g.*, a knitting needle) longitudinally through them.

A fractured sternum with displacement of the parts should be plated. A fracture of the scapula is treated by fixing the arm to the chest. In compound comminuted fractures the edges of the wound should be excised, the fragments of bone removed and the wound closed by sutures either immediately or after a delay of forty-eight hours.

Treatment of Pleurisy.

The treatment of the pain of dry pleurisy by separating the visceral from the parietal pleura by oxygen, so as to prevent the friction of the two membranes on each other, has already been dealt with on p. 39. Pleural effusions have been discussed on pp. 46 *et seq.*

Treatment of the Symptoms consequent on Compression of the Chest.

Traumatic asphyxia. Shock is the only symptom of this condition which calls for treatment. The discolouration due to the extravasated blood from the ruptured capillaries and the œdema need no active intervention. The blood and the fluid are absorbed in the course of a few days.

Rupture of a bronchus: Surgical emphysema. A ruptured bronchus must be exposed by operation and sutured. The technique of this is described in Chapter VI. The injury is usually complicated by mediastinal emphysema, the consequences of which are often so serious as to preclude the possibility of saving life by operation. Tiegel,* as a preliminary to the operation for closing the wound in the bronchus,† recommends that an incision should be made through the tissues immediately above the manubrium sterni and that a Bier's cupping glass, connected with some form of pump to exhaust the air, should be fixed over the incision so as to aspirate some of the gas from the mediastinal tissues and relieve the pressure.

* "Mediastinalemphysem." Centralblatt fur Chirurgie. 1911.

† The technique for the exposure of and closure of the bronchus is the same as that described on p. 120, Chapter VI.

Sauerbruch does the operation for suture of the bronchus in a hypo-atmospheric chamber and by this means some of the gas in the mediastinum is sucked out during the course of the operation.

Mediastinal emphysema which occurs owing to rupture of the pleura, as a complication of pneumothorax, must be treated on the same principles as have already been laid down for the relief of ingravescent pneumothorax. (See p. 84.)

In subcutaneous emphysema, surgical intervention is needed only when the distension of the skin is so great that there is danger of sloughing. A few incisions through the skin and subcutaneous tissues will relieve the tension.

Treatment of Contusions of the Lung.

The nursing of patients suffering from this injury is the most important part of the treatment. The danger of the possibility of secondary infection of the lung from organisms in the upper respiratory track must be constantly borne in mind. This danger can be minimised if, in addition to fresh air and healthy surroundings, the hygiene of the mouth and throat is given every attention.

Treatment of Massive Collapse.

It has been stated earlier in the chapter that all symptoms of massive collapse are due to the sudden and extreme diminution in the volume of the lung, in consequence of which there is a high intrapleural negative pressure and marked anatomical-mechanical changes. It has already been shown in Chapters II and IV that we possess the means of controlling absolutely intrathoracic pressures. It is obvious, therefore, that if these means are applied in cases of massive collapse all the symptoms can be entirely abolished. The control should be effected by oxygen, and sufficient of this gas should be run into the pleural cavity to neutralise the increase in the negative pressure due to the diminution in the volume of the lung. The technique of this has been described on p. 39 and pp. 164 *et seq.*

Treatment of Hæmothorax.

A.—*Simple aseptic hæmothorax of recent origin.* In the previous chapter considerable emphasis has been laid on the necessity for the early and complete removal of pleural effusions, the reason for this being the interference with the free expansion of the lung when there has been any deposit of fibrin on the pleural membranes. In a hæmothorax, the deposit of fibrin from the blood is great and it may be increased by the secondary pleural exudate, so that for this reason alone, and quite independently of other causes such as delayed absorption, it is essential that the blood should be removed as soon as possible. It used to be maintained that if the removal of blood was attempted within the first week or two of the injury, there

would be a recurrence of the hæmorrhage, the clot occluding the damaged vessel or vessels being separated by the pull of the negative pressure. With the modern method of treatment (oxygen replacement), which allows of complete control of the intrapleural pressure, such danger does not exist. It has, moreover, been found that after the first three or four days the pressure in the pleural cavity can be made slightly negative without fear of a recurrence of the bleeding. The treatment, therefore, that should be adopted in all cases of simple hæmothorax is the complete removal, after the third day, of the whole of the blood by oxygen replacement.* The pressure left in the pleural cavity should be equal to +1 mm. of mercury if the aspiration is done within the first week after the injury; after that, a negative pressure equal to 5 or 7 mm. of mercury can be left with safety. It will sometimes be found that in the course of the next few days, a small pleural effusion has developed; this should be removed and the gas in the pleural cavity changed by oxygen replacement. On this occasion a considerably higher intrapleural negative pressure is left (equal to 12 or 15 mm. of mercury, if the patient can tolerate this without pain).

B.—*Incomplete absorption.* In some cases of aseptic hæmothorax which have been left untreated, the fluid contents of the blood become absorbed and the deposited fibrin is in part absorbed and in part organised. The lung re-expands, the visceral and parietal pleural membranes again come into contact and the functional capacity of the lung is in a considerable measure restored. There is, however, always a residuum of symptoms. In some cases these are continuously present, causing a constant though a slight interference with the patient's capacity for the enjoyment of life. In others, the symptoms due to incomplete absorption come into evidence on exertion only. They are all due to the same cause, viz., the interference with the normal physiological movements of the lung, the thickened pleura checking the free expansion of the organs and disturbing thereby the synchronous movements of the lungs and of the chest wall.

Absorption is, however, by no means a constant process and in many cases the pleural cavity remains filled with the altered blood, part gelatinous, part fluid. A patient with a hæmothorax which has failed to absorb is ill. He has little enjoyment of life, he is unable to exert himself, is easily tired, his appetite is affected and there is often a moderate fever. Aspiration is impossible; a few c.c. of fluid may be withdrawn, but the needle is quickly obstructed by the gelatinous clot. If the hæmothorax is left untreated the patient will become a chronic invalid. The whole of the blood must be removed through an incision in the chest wall. The effect of this on the patient and the improvement in his general condition is remarkable and is immediate. An opening is made through the lower part of the chest wall, a length of rib being resected for this purpose as in the operation for empyema.

* The technique is described on p. 46.

The fluid part of the blood drains away, and the solid part is detached by gently mopping over the surfaces of the pleura with gauze, removing at the same time not only the fibrin but also any mass of clot that may be present. The wound is then completely closed by sutures and the air in the pleural cavity is replaced by oxygen. The intrapleural pressure at the end of this operation should be equal to about -12 mm. of mercury. The details of this operation and the after treatment are the same as those described on pp. 59 and 60.

C.—*Infected hæmothorax.* The treatment of an infected hæmothorax differs in no way from that of an empyema (see p. 70). Especial care must be taken that the whole of the clot is removed at the time of the

Fig. 34. Bullet lodged in the right lung five years previously. Tuberculosis of the right upper and middle and of the left upper and lower lobes. At the autopsy, the bullet was found to be projecting into the lumen of the large cavity seen in the radiogram.

operation, together with any metal fragment which may be lodged in the pleural cavity. Shell fragments, owing to the irregularity of their surfaces, become lodged in the tissues ; shrapnel bullets, on the other hand, usually drop to the bottom of the pleural cavity and become embedded in the debris between the diaphragm and the lateral and posterior aspects of the chest wall.

The method of treatment just indicated may be considerably modified in certain cases. The experience gained in war surgery has shown us that if

the tissues of the body are given the opportunity, they are quite competent to deal efficiently with infective processes provided that the toxic changes due to the organisms are not too virulent. Two essentials for the success of the resistance of the tissues are, the absence of a nidus of inert matter (*e.g.*, blood-clot) and of lacerated devitalised tissue in which the organisms can flourish. The second essential is that the tissues shall be safeguarded from secondary infection. This can only be done by the closure of the wound. It has frequently been found possible to obtain even primary union by completely excising the margins of a lacerated wound, removing all debris and approximating by sutures the whole of the raw areas. There is one part of this technique which it is difficult and often impossible to obtain when treating intrathoracic injuries ; it is extremely difficult completely to obliterate the cavity between the lung and the chest wall, and as a result of this there is always a potential source of failure. It has been found, however, that in practice convalescence can be greatly shortened if this line of treatment is applied. In all cases, therefore, of hæmothorax in which, from the symptoms, there is strong suspicion that the blood clot is being pervaded by organisms, the chest should be opened, the whole clot removed with as little disintegration as possible, the pleural cavity thoroughly mopped out and the wound completely closed. Immediate closure of the wound should also be tried in cases of pyothorax in which the infection is mild in character. It is always an advantage after suture to replace the air in the pleural cavity by oxygen at a slight negative pressure. Failure to arrest the infective processes will not have increased the danger to the patient as the sutures in the wound can be easily removed and a drainage tube inserted at the first indication of septic absorption.

Treatment of Pneumo- and Hæmo-pneumothorax.

The principles of the treatment of gas in the pleural cavity, whether alone or in conjunction with a liquid, and of the treatment of the ingravescent type of pneumothorax have already been fully detailed on pp. 62 and 84.

Treatment of Injuries and Lacerations of the Lung and of Uncontrolled Hæmorrhage.

The seriousness of all cases of trauma of the lung where there has been laceration due to compression or to a penetrating wound made by a foreign body, is that some portions of the lung tissue are actually destroyed, while the vitality of other parts is reduced by interference with the blood supply. The injured lung tissue is, moreover, frequently contaminated at the time of injury and a primary or else a secondary infection is a frequent result. Since infection of the lung parenchyma or of the pleural cavity is the gravest complication of trauma, it is obviously of the greatest importance that every available means should be utilised to diminish the risk of its occurrence or the virulence and extent of the sepsis when it has occurred.

Experience has shown that early and energetic measures are the most beneficial in counteracting these dangers and with many surgeons, therefore, operative intervention is the routine treatment of the more serious cases of trauma. The damage caused to the lung tissue by a sharp instrument or a perforating bullet is comparatively slight, and the only indication for operation on this type of wound is the presence of a complication such as persistent bleeding or injury to a bronchus. Wounds due to irregular shell fragments are, however, particularly dangerous, not only on account of the damage to the lung parenchyma, but because of the frequency with which the metal carries into the substance of the lung fragments of clothing or fragments of bone. Intermediate between these two types of injury come those cases in which there is penetration by multiple small fragments. The difficulty of tracing out such wounds and of finding the scattered foreign bodies are so great as scarcely to justify intervention. The majority of these cases, moreover, after a preliminary pneumonic consolidation of the implicated area, recover completely ; occasionally only does local infection result.

The danger of infection is not the only reason why operative treatment is advisable. The sequelæ, as well as the complications, may have a most deleterious influence on the patient's future existence. The most important of these after changes is that of bronchial dilatation, secondary to the fibrosis of the lung, which is in reality a consequence of infection. The mechanism by which this bronchial dilatation develops is dealt with in Chapter IX. Another sequela is that of repeated hæmoptysis due to the constant irritation caused by the foreign body. This symptom is rarely dangerous nor does it incapacitate the patient ; but the constant repetition of it is disturbing to his mental state.

Shock is the most serious contra-indication to operative intervention, and for this reason active treatment must always be postponed a few hours after the injury to allow time for the acute stage of the shock to subside. It has been found that fairly extensive intrapleural manipulations can be carried out without the aid of apparatus for compensating the exposure of the lung surface to atmospheric pressure ; provided always that some degree of traction is maintained on the organ to check the to and fro flapping of the mediastinum. Intratracheal insufflation, when available, does undoubtedly allow of freer manipulation ; but the necessity of administering a general anæsthetic instead of a local or regional one may counterbalance this advantage. The simplest route of access is by a long incision through an intercostal space. As soon as the pleural cavity has been opened it must be cleansed, so as to allow a clear view of the seat and extent of the lung lesion. Lacerated fragments of the lung are cut away, and a badly torn wound of the lung should be completely excised. A metal or bony foreign body is easily localised by palpation, unless the operation has been postponed until pneumonic consolidation has occurred. The foreign body and pieces of clothing carried in with it are removed, and the track of the missile is

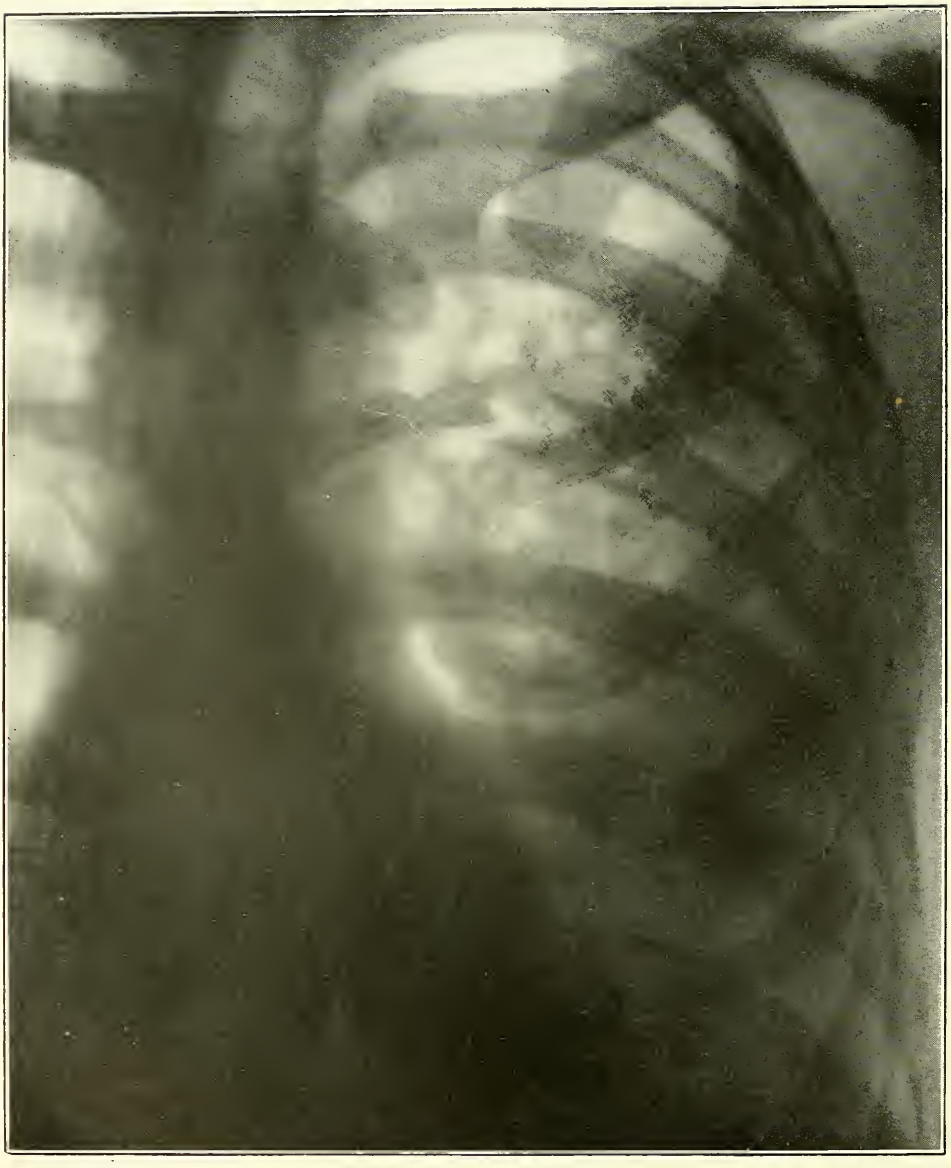

Fig. 35.

Gunshot wound of the left lung. The metal fragments in the upper lobe are surrounded by areas of intense infiltration. There is pneumonia of the lower lobe. The anterior part of the 2nd rib and the posterior part of the 6th rib have been fractured.

cleansed with gauze. The wound in the lung or the raw surfaces caused by excision of lacerated tissue is closed by catgut sutures, and the thoracic wound is sutured and made airtight. Finally the air in the pleural cavity is replaced by oxygen at a negative pressure.

The treatment of *continuous bleeding* in association with laceration of the lung is identical with that described above, except that if the blood pressure is steadily decreasing any delay is dangerous.

Treatment of Injuries causing an Open Wound in the Chest Wall.

The danger of this has already been discussed on p. 104. Immediate steps must be taken to reduce the aperture so as to control the passage of air. Until the preparations for operative treatment are completed, the wound must be kept covered by a large dressing firmly applied over it. Treatment consists in cleaning out the pleural cavity, in repairing the damage to lung tissue, and then in obliterating the opening in the chest wall by some form of plastic operation. Even though suppuration is almost certain to follow, it is advisable at this early stage to devise a flap which will completely occlude the opening, and to suture the margins so that the wound is as far as possible airtight. The opening is probably not in the position which would allow efficient drainage of an infected pleural cavity. When, therefore, and as soon as the pyothorax develops, this should be drained through a separate incision opening into the lowest part of the cavity.

Treatment of Associated Lesions.

Special attention must be called to injuries of the pericardium and to the diaphragm. A rent in the diaphragm must be stitched up at the time of treatment of the lung. A pericardial effusion may complicate any of the lesions described above; it is usually simple but may suppurate. Such an effusion, if it embarrasses the heart's action, must be syphoned off through a small incision.

Rupture of the diaphragm increases the danger of the spread of infection from the abdomen, and there is always the risk of the escape of part of the abdominal contents into the thoracic cavity, and of strangulation. Such rents must, therefore, be occluded by a series of mattress sutures inserted at the time of treatment of the damaged lung.

HERNIA OF THE LUNG.

This condition is due to rupture of the pleura. The hernia may consist of a bulging of the lung into the subcutaneous tissues only, but it is often complete or compound, part of the lung being forced by a violent expiratory effort through a wound in the chest wall. This latter condition occurs as the result of wounds caused by some penetrating instrument, whilst the former

is usually due to a blow. Treatment of a subcutaneous hernia which can be reduced consists in the application of a pad applied firmly over the weak spot in the chest wall and maintained there for three or four weeks. The lung becomes adherent to the surrounding tissues; this and the fibrotic changes in the wound are usually sufficient to prevent further protrusion. Should this fail, a plastic operation will be necessary. The defect in the chest wall is made good by splitting the adjacent ribs or cartilages and displacing the superficial portions across the gap.

A hernia which projects through the thickness of the chest wall is in most cases gripped by the intercostal muscles and, if left, becomes strangulated. The treatment depends on whether the hernia is seen before or after the strangulation has occurred. In the former case, the colour is normal, and the lung tissue crepitates on pressure. The surface of the protruding mass must be carefully cleaned, grasped with a piece of gauze and an attempt made at reduction; it may be necessary to divide the constriction at the neck of the hernia. When the reduction has been effected, the wound in the parietes is sutured. A hernia, when strangulated, must be cut away with a knife or cautery and the wound allowed to heal by granulations. The lung is usually fixed by adhesions round the neck of the sac, but to ensure against the retraction of the organ, and the development of an empyema from the infected stump, it is advisable, before amputating the hernia, to fix the lung to the chest wall round the wound by a few catgut stitches.

DIAPHRAGMATIC HERNIA.

A diaphragmatic hernia is either a congenital condition due to imperfect development of the diaphragm, or is acquired. The acquired herniæ are the result of injury (a violent blow or stab) or of the yielding of a congenitally weak diaphragm to violent intra-abdominal pressure, as in whooping cough. The hernia occurs most frequently into the left side of the chest. It may contain any of the suprapelvic abdominal organs, but those most commonly found are the stomach and the colon, next in order of frequency come omentum and small intestine, and then liver, duodenum, pancreas and cæcum. In the majority of cases the hernia is a false one, that is to say, it has no peritoneal sac.

As a natural corollary of the displacement of the intra-abdominal organs, their normal function is interfered with, this being in some measure proportionate to the size of the hernia. There is also considerable disturbance of the intrathoracic organs, displacement of the heart and of the lung in acquired herniæ. When the hernia is congenital, the lung on that side is partly or entirely atelectatic.

The clinical picture combines abdominal and thoracic symptoms—indigestion, flatulence, constipation, diarrhœa and pain associated with food. The thoracic symptoms are shortness of breath and palpitation.

The signs are suggestive of a basal pneumothorax, that is to say, there is a tympanitic note on percussion and absence of breath sounds ; but over the lower or even the greater part of one side of the chest a stomach splash and the gurglings due to intestinal peristalsis may be heard. The physical signs of the lung above the hernia are normal or those characteristic of a

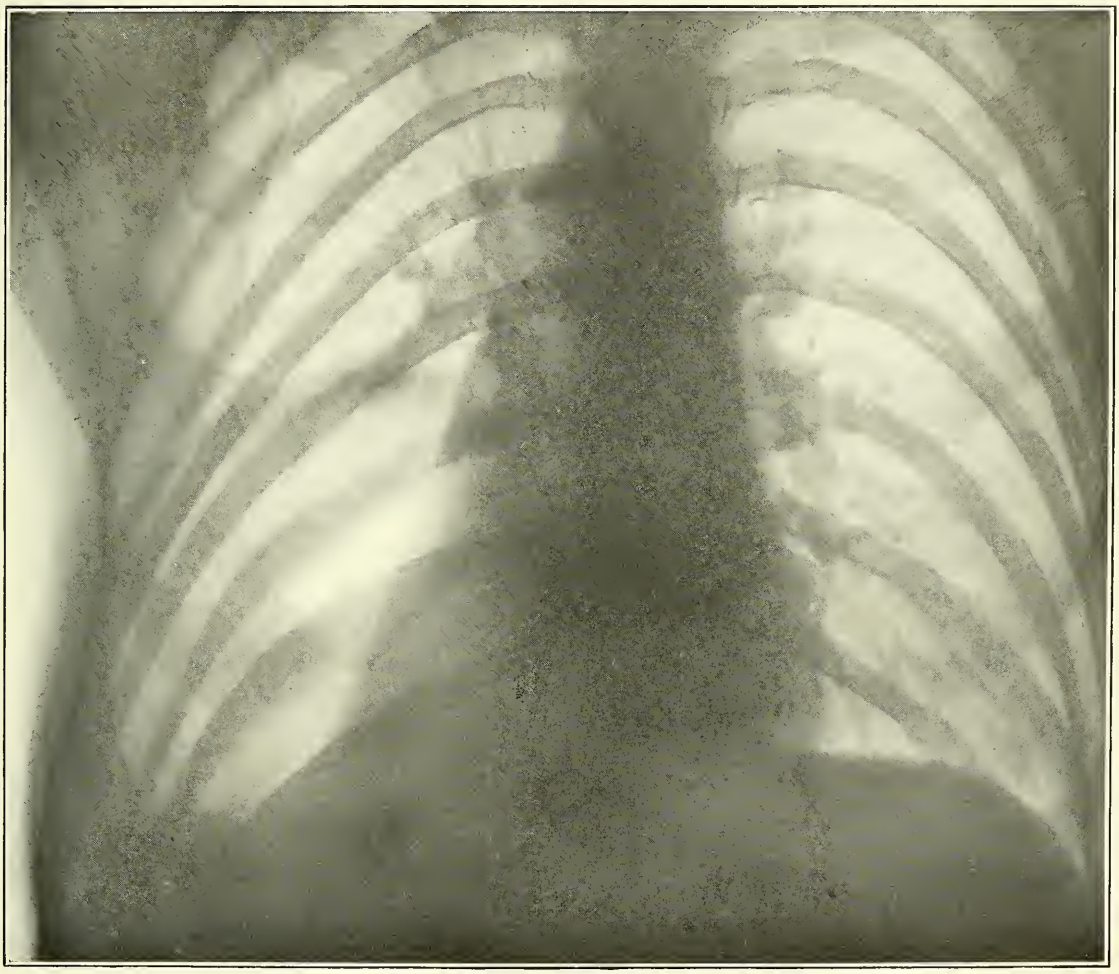

Fig. 36. Diaphragmatic hernia. The diaphragm on the right side is well defined, but on the left it is extremely ill defined. The left lung is displaced upwards by the stomach, the upper margin of which appears as a faint dome-shaped shadow.

compressed or atelectatic lung. This clinical picture may be further confused by the presence of disease in the herniated organs or of strangulation.

The X-ray appearances are characteristic. The outline of the diaphragm is ill-defined and imperfect. Above, occupying the position of the base

of the lung, there is, when the stomach only is herniated, a clear dome-like area with an extremely thin margin ; but when the colon is included, there are shadows indicative of that organ. If any doubt exists as to what part of the intestine is occupying the hernia, this can be settled by noting the passage of a bismuth meal.

In my case (Fig. 36) the peristaltic movements of the stomach could be watched on the screen ; there was a shadow which gradually appeared, traversed the clear area and then disappeared, regularly once every eight seconds.

TREATMENT.

Operative intervention should be reserved for those cases only in which the symptoms are distressing and incapacitate the patient, as the operation is a difficult one and the success of it problematical. The object of the operation must be to replace the abdominal organs and to close the opening in the diaphragm. The best route of access is by an abdomino-thoracic incision extending upwards along the outer border of the rectus muscle through the costal margin, and upwards, outwards and backwards along the 8th intercostal space. In the absence of a peritoneal sac, the abdominal organs must be dissected free from the margins of the diaphragm. When replacement has been effected, the opening in the diaphragm is closed either by suture of the separated margins, by decostalising the adjacent portion of the chest wall and approximating it to the diaphragm, or by closing the defect in the diaphragm by a graft of fascia lata.

Chapter VI.

FOREIGN BODIES IN THE BRONCHI.

Foreign bodies enter the bronchi usually by inhalation, but, rarely, also by ulcerating through from the œsophagus or by penetrating through the chest wall. The point of arrest of the inhaled foreign body depends to some extent upon its size and shape. It lodges usually in the right bronchus, as the lumen of this is in almost a direct line with that of the trachea. When a smooth rounded substance has been inhaled, it may remain loose and be kept in constant movement by attacks of coughing. The symptoms are then most distressing; severe and constant cough and dyspnœa, vomiting, suffocation, unconsciousness and even death if the body is driven up to and impacted in the glottis.

At the moment of aspiration, the symptoms may be of any degree of severity from an attack of coughing to death by suffocation.* The subsequent history depends on the character of the inhaled substance; particularly on the regularity or irregularity of the outline, on its capacity to absorb moisture and thereby to increase in size, and on the presence or absence of infection. Grains of corn and sharp-pointed substances often penetrate through the wall of the bronchus and traverse the lung or mediastinum.

The pathological changes and, consequently, the clinical phenomena also are divisible into those which occur during the first few weeks and those which develop at a later date. A foreign body produces irritation of the tissues around it; there is, for a time at any rate, spasm of the bronchus, swelling of the walls, and an outpouring of secretion. The partial or complete occlusion of the air-way to that part of the lung in communication with the obstructed bronchus depends on the intensity of these changes and on the size, the shape and the character of the inhaled substance. Complete obstruction is generally due to bodies fairly uniform in diameter, or to substances like food which swell from the absorption of moisture. The air in the alveoli beyond a completely occluded bronchus is absorbed, and

* If the foreign body is inhaled during an anæsthetic the symptoms may be so slight as to escape notice.

that part of the lung shrinks into a condition of atelectasis. When, however, the obstruction in the bronchus is partial only, the air enters the lung more easily than it escapes from it, and a condition of over-distension of the alveoli is for a time established. In the former case there is retraction of the chest, dullness on percussion and absence of breath sounds ; in the latter, the chest wall is expanded, the diaphragm is displaced downwards, the percussion note is tympanitic, while the breath sounds are weak.* Over distension of the lung is a temporary condition only. With increase in the secretion and of the changes in the walls of the bronchus, the alveoli become blocked, air entry is further interfered with, and the air in the lung is absorbed.

If the foreign body is septic or undergoes decomposition, it will cause septic pneumonia, abscess or gangrene, empyema, putrid bronchitis or even mediastinal suppuration. Local suppuration, ending possibly in perforation, gangrene or secondary hæmorrhage, occurs mostly with sharp irregular-shaped bodies. On the other hand, foreign bodies which are fairly clean and not liable to decomposition may become encapsuled and cause no special symptoms (except possibly slight bronchitis) for weeks, months or even many years.

Later manifestations are due to chronic but progressive fibrotic changes associated with the chronic irritation of the bronchus and adjacent lung parenchyma, or resulting from the incomplete resolution of a pneumonic lung or of a lobe which has become atelectatic. The influence of these fibrotic changes is seen in the dilatation of the bronchi and the development of all the symptoms and signs of bronchiectasis.

Occasionally a foreign body, such as a grain of corn which has been symptomless for months or years, will reach some part of the surface of the body and form a subcutaneous abscess. The lung or the track of the grain may be infected by streptotrichosis. Rarely, the organisms which have been enclosed in the capsule formed round some foreign body increase suddenly in number and in virulence and form an abscess. In such cases it sometimes happens that when the abscess bursts into a bronchus, the original cause of the trouble, which has become loosened by the suppuration, is expelled with the pus ; spontaneous healing may follow if the abscess is small and free drainage into the bronchus has been established.

TREATMENT.

The complications, suppuration and bronchiectasis, of foreign bodies in the bronchi are so serious that it is obvious that every effort must be made to extract the body within as short an interval of time as possible.

* The condition is often erroneously diagnosed as a pneumothorax.

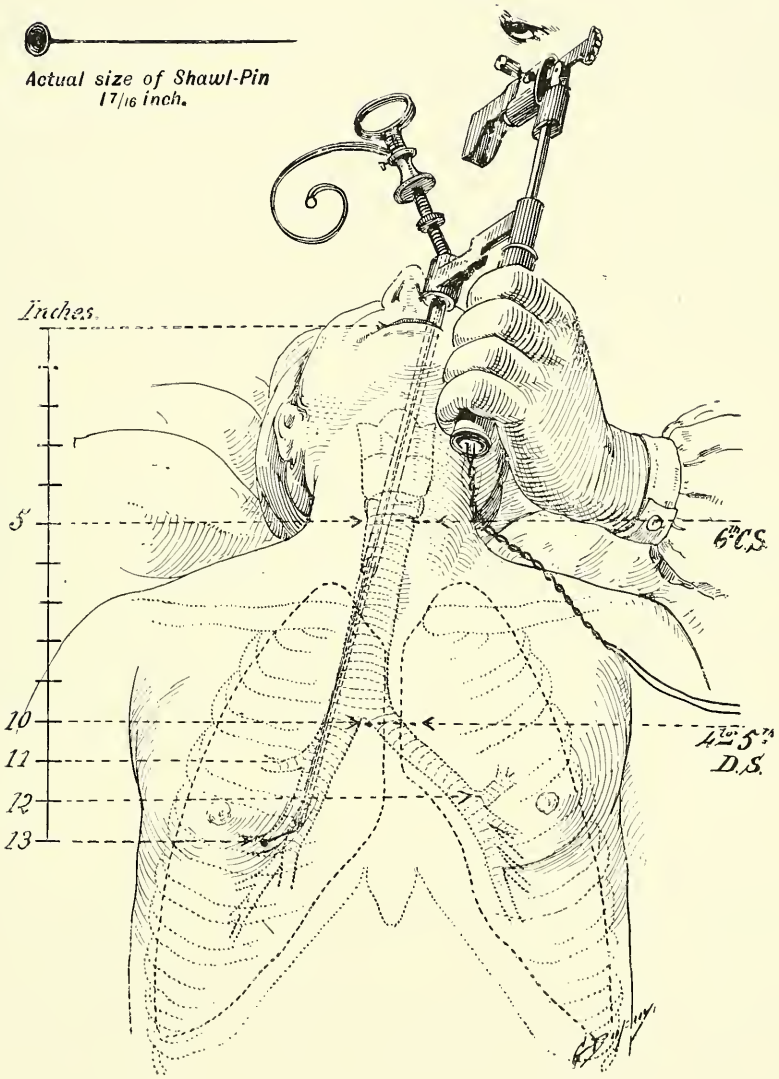

Fig. 37. Direct Bronchoscopy (from "Diseases of the Nose and Throat," Sir St. Clair Thomson (Cassell & Co.)).

Occasionally, immediate operation is necessary to save life ; this is particularly the case when suffocation threatens directly on inhalation as the result of impaction in the glottis, or on account of the spasmodic closure of the glottis owing to the foreign body being continually driven against it by the coughing efforts at expulsion. Laryngotomy or tracheotomy must be done immediately ; this will enable the patient to recover from the asphyxiation ; the foreign body, if movable, may then be spontaneously expelled, or if impacted, it can be dealt with at leisure.

A foreign body must be accurately localised by radiography before any attempt at removal is made. This is, of course, not possible when the inhaled substance is translucent to the X-rays. Transpleural or transmediastinal operations are dangerous to life and often unsuccessful ; they must therefore never be attempted unless the foreign body is inaccessible for removal by bronchoscopy and can be localised by the X-rays.

BRONCHOSCOPY.

The bronchoscope, described very briefly,* consists of a strong rigid handle, at right angles to which are two supports. The upper has hinged on it the shaded electric lamp and the adjustable reflecting mirror and eye-piece ; the lower is for the attachment of the laryngeal spatula. An accurately fitting extending bronchoscopic tube, which is passed through the laryngeal spatula when *in situ* in the larynx, enables the operator to examine the primary and also the secondary bronchi, and, by means of special forceps, to manipulate and extract foreign bodies from these depths in the air passage.

TRANSMEDIASTINAL BRONCHOTOMY.†

The patient is anæsthetised by the intratracheal method and is placed and supported on the table, resting on the affected side with the lower shoulder drawn well forwards. A double flap is turned outwards (*i.e.*, downwards towards the table when the patient is lying on the affected side), the first layer consisting of skin and superficial muscles, and the second of ribs and intercostal muscles The base of the flap lies along the vertebral border of the scapula, and the opposite free edges along the tips of the

* For all details of the instrument, methods of anæsthetisation, position of the patient for examination, methods of introduction and uses of the instrument, the reader is referred to Brunig's "Handbuch für die Tecknik und Methodik der direckten Okulären Methoden," translated and edited by Howarth.

† The transpleural route of access to the bronchi, owing to the danger of infecting the pleural cavity by the extremely septic mucus from the bronchial tube, should not be used.

transverse processes ; the ribs reflected in the deep flap are the posterior parts of the 5th to the 8th inclusive.* The pleura and lung are displaced outwards and forwards by blunt dissection. On the right side, the œsophagus, a branch of the vena azygos, the vagus nerve and the right bronchus are exposed (*vide* Fig. 8) ; and on the left, the aorta, vagus and left bronchus. Before proceeding further with the operation, the vagus nerve should be anæsthetised as high as possible with 2 per cent. novocaine.† The bronchus is opened longitudinally‡ and an attempt made to find and remove the foreign body with forceps. Tiegel recommends that the opening in the bronchus be closed by a series of interrupted catgut sutures inserted as shown in Fig. 38. This line of sutures is then covered by the overlying pleura which is drawn back and stitched over it. The wound is closed by replacing the two flaps, catgut being used for the deep sutures.

Drainage of the wound is very important owing to the probability of infection from the mucus of the bronchus ; but drainage is also dangerous,

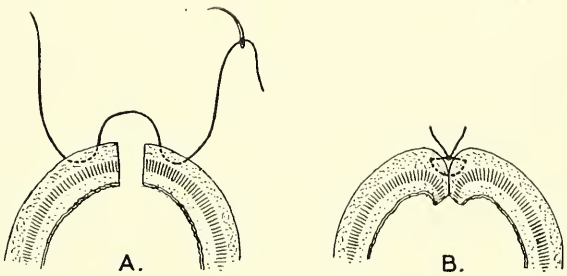

Fig. 38. Suture recommended for the closure of a wound in the bronchus. (After Tiegel.)

as air may be sucked in through the tube and cause an extrapleural pneumothorax and mediastinal emphysema. To minimise this danger the tube must be small and well covered with dressings ; the tube should be clamped when these are changed.

The treatment of the complications of foreign bodies (*e.g.*, abscess and bronchiectasis) is discussed in the chapters dealing with these diseases.

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* For technique of cutting these flaps, see p. 88.

† As the result of experimental work, I have found that manipulations of the lung, especially in the region of the hilum, are tolerated with much less shock if the vagus is previously anæsthetised.

‡ Before opening the bronchus, the adjacent parts of the wound should be packed with gauze soaked in some antiseptic solution.

Broncholiths.

Broncholiths are calcareous concretions formed in cavities of the lung, either phthisical or bronchiectatic, in which there is prolonged stagnation of the secretions. They are rarely found except in cases of chronic phthisis. These broncholiths are always small and usually very irregular in outline. On account of their sharpness, they may, when expelled from the cavity, cause pain and bleeding from injury to the mucous membrane of the bronchus and trachea. They are opaque to the X-rays (see Fig. 44). They are of interest radiologically, but they produce no symptoms except, occasionally, slight laceration of the mucous membrane of the trachea when they have been dislodged from the cavity and are expelled with the sputum.

Chapter VII.

GANGRENE AND ABSCESS.

The various acute and chronic suppurative processes in the lung are so closely allied that it is often impossible to determine by clinical means the nature of the morbid changes. The authorities on this subject differ greatly as to what constitutes a gangrene as opposed to an acute abscess. By some, every form of suppuration with offensive expectoration is labelled gangrene, and a cavity containing non-fœtid pus is referred to as an abscess; others again differentiate between gangrene with odourless or gangrene with stinking pus. It is necessary, therefore, to define what conditions will be described in this book under the terms "gangrene" and "abscess" respectively.

A focus of acute suppuration developing in a lobe of the lung is surrounded by an area of pneumonic consolidation, and there is no sharp line of demarcation between the diseased and healthy tissue. The lung parenchyma in the affected zone breaks down and the disintegration may be of any grade from molecular to massive. In the former case the result is a debris only among the accumulating pus cells; but in the latter case there is a separation of fragments of necrosed tissue sufficient in size to be recognisable in the fluid. The infected area does not, at first, necessarily open into a bronchus, but communication eventually occurs and turbid fluid or pus escapes. The subsequent course depends on the severity of the infection and on the freedom of the drainage. The acute suppurative process may continue, the rate of spread and disintegration being such as to preclude the possibility of any line of defence being formed; or the drainage may be efficient in checking the progress without curing it, when a surrounding wall of fibrous tissue will be established and the infective processes will become localised into a chronic abscess with well-defined walls and an encircling zone of consolidated lung. Rarely, and as a rule only when the focus is in the upper lobe, is the drainage through the bronchus sufficient to establish a cure.

An offensive odour of the sputum and of the breath may be due to the presence of saprophytic organisms, to the retention of secretions or to necrosis; a fœtid stench can be caused only by a massive and rapid necrosis of the tissues. There can be no doubt that the use of the term "gangrene" is correct when the breath is fœtid and the sputum is stinking, is dark in colour owing to disintegrated blood, and contains obvious pieces of necrosed tissue. But it is much more difficult to establish a well-defined clinical

distinction between the cases of acute suppuration with necrosis, in which the symptoms due to this morbid process are not so pronounced, and those cases without actual necrosis.* The treatment, moreover, of all these acute changes is the same. For these reasons, all cases of acute suppuration in the lung parenchyma will be grouped in this chapter under the term "gangrene," and all cases of chronic suppuration under the term "chronic abscess."

Just as in the acute suppurative processes it is difficult to determine by clinical means the exact nature of the morbid changes, so too is it at times impossible to decide whether a chronic abscess cavity associated with bronchial dilatation is due to a chronic abscess with secondary bronchiectasis, a primary bronchiectasis with a secondary abscess in the lung parenchyma, or a bronchiectasis complicated by a greatly dilated saccule projecting from the side of a bronchial tube into the lung substance. The clinical and radiological resemblance which may exist between these three types of lesion is at times so close that in actual practice a differentiation cannot be made,† and certain forms of bronchiectasis might well, therefore, be considered in conjunction with chronic abscess. On the other hand, the treatment of brochiectasis, of whatever type, differs radically from that of chronic abscess uncomplicated by bronchial dilatation, and for this reason the whole subject of bronchiectasis is discussed in a separate chapter.

There are three chief factors which determine the onset of suppuration of the lung. They are (1) the entry into the lung of infective organisms; (2) the presence of some morbid change in the lung substance such as may be occasioned by the inhalation of irritant substances (anæsthetics, poison gas), by a contusion of the lung by injury, or by invasion of the parenchyma by non-pyogenic organisms (viz., pneumococcus, bacillus of influenza); (3) the lowering of the general resistance of the tissues, especially by the presence of some disease such as diabetes.

The pyogenic organisms may reach the lung by the blood stream, as in cases of septicæmia or pyæmia. Their entry may be through the bronchi and occurs in this way most frequently as a result of aspiration, while the patient is under an anæsthetic, of infective substances such as mucus, a piece of tooth or tartar or part of a growth (*e.g.*, adenoids or ulcerated carcinoma). Occasionally the organisms are carried in with some foreign body accidentally inhaled, such as a grain of corn. But infection may also spread from the upper air passages to a damaged part of the lung without any such gross method of conveyance as mentioned above; there is, however, usually some considerable focus of origin such as pyorrhœa alveolaris. A third route of entry is by direct invasion either by the rupture into the lung of pus from the pleural cavity or from a sub-diaphragmatic abscess, or by

* The symptoms and signs of acute suppuration in the lung, associated with the expectoration of a non-fœtid sputum of a yellow or greenish colour, are usually due to the rupture into a bronchus of an encysted pleural or interlobar empyema.

† Elastic tissue in the sputum is an indication that the lung parenchyma is involved.

ulceration from a growth in the œsophagus. Finally the lung substance may be infected by the direct implantation of organisms by bodies perforating the chest wall (*e.g.*, gunshot wounds).

It is impossible to foretell exactly what combination of circumstances is necessary to enable a contamination to become an infection or to determine that the infection shall develop into an acute suppuration with necrosis of the tissues, or remain limited as a chronic abscess. It undoubtedly depends on the extent of the influence of the three primary factors, and possibly more on the virulence of the organisms than on either of the other two. As a general rule, it may be said that acute suppuration or gangrene always follows direct invasion of the lung from without (*e.g.*, rupture of a pyothorax), and from within *via* the blood stream (*e.g.*, septicæmia or septic emboli*). The inhalation of organisms into lung tissue, the power of resistance of which has been diminished either by irritation or by injury, leads usually to acute suppuration, but the infection may be sub-acute in character or even remain latent. In the latter case, when symptoms of a chronic abscess finally become definite, the interval of time which has elapsed between the cause and its effect may be so considerable that the relationship of the two and the importance of the past history is unappreciated by the patient.

The clinical manifestations of a chronic abscess may from the start be those of a chronic infection, but frequently an abscess is a sequela of gangrene in a patient who has survived the acute stages. The spread of suppuration into the parenchyma from an infective condition of the lung such as bronchiectasis is usually of the nature of a chronic abscess rather than gangrene.†

If the opening of communication, and therefore of drainage, from an abscess into a bronchus become obstructed, the virulence of the organisms will increase and a chronic will become converted into an acute suppurative process, which may result in erosion of the surrounding walls and a gangrenous extension into the adjacent lung tissue.

In gangrene the affected part of the lung is intensely congested and is in a state of hepatization. In the centre of this there is an area of necrosis, the tissues changing to greenish or greyish black and being extremely friable. Gangrene may spread rapidly without the formation of any actual cavity; later there is liquefaction of the tissues and communication with a bronchus is established. In the central part of the infected area there is then a small space bounded by extremely irregular and sloughing walls. If, as the result of free drainage through a bronchus, the virulence of the organisms subsides, the living tissues may be able to react and to establish a protecting boundary.

* The source of the septic embolus is usually an otitis media with lateral sinus thrombosis, or an infective thrombus of the lower limbs or pelvis.

† Chronic abscess cavities are frequently present in pulmonary tuberculosis; they are dealt with in Chapter VIII. Hydatid cysts which have become secondarily infected are discussed in Chapter XI.

This eventually becomes the fibrous wall of the chronic abscess. Should there be a recrudescence of the virulence of the organisms, this wall will in turn be destroyed and the acute process breaking through, invade the adjacent lung parenchyma. A gangrenous focus is at first usually single, but secondary foci may develop round it. Whilst in the chronic stage the vessels in the immediate neighbourhood are thrombosed, in the acute lesion this obliteration does not take place. In the latter case, therefore, hæmorrhage into the tissues during the separation of a sphacelus, and hæmoptysis when there is a communication into the bronchus, are constantly present. If the suppuration approaches the surface of the lung, it sets up inflammatory changes in the pleura, causing pleurisy with adhesions, pleurisy with effusion or empyema.

The variety of organisms found is very great. The most important of the pathogenic group are the streptococcus, staphylococcus and the bacillus coli; of the saprophytic group, which are responsible for the putrefactive processes, the commonest are the proteus and tetragenus.

SYMPTOMS AND SIGNS OF GANGRENE AND CHRONIC ABSCESS.

The constitutional disturbance, the fever, together with the increase in the rate of the respiration and of the pulse, and sometimes the odour of the breath, are often the first indications of acute gangrene. When, however, the suppurative processes develop in a lung already diseased, the onset of gangrene will be marked by an exacerbation of existing symptoms rather than by the development of new ones (the odour of the breath, when present, is an exception). A deep-seated focus of suppuration will often give symptoms for some days before any definite signs can be discovered; this was the case in the patient whose radiogram is shown in Fig. 39. Her general condition and the odour of her breath led to the correct diagnosis, but no physical sign indicating the situation of the gangrene could be found for three days, when the gangrene ruptured into a bronchus and the signs of a cavity developed rapidly. Dyspnœa is proportionate to the rate of increase and to the extent of the gangrene; in chronic abscess it is constantly present but is not intense. Pain is due, in most cases, to the involvement of the parietal pleura. Cough and expectoration are constant symptoms of both acute and chronic lesions when there is communication into a bronchus. The cough may be present even when there is no such connection; it is then associated with a mucoid expectoration.

The sputum is purulent in both gangrene and chronic abscess, but in the former condition it has at first a watery consistency and is dark red or brown in colour (even at times bright red), owing to the presence of blood, and may contain fragments of necrosed lung tissue. In both cases elastic fibres, often retaining the shape of the alveoli, can be demonstrated with the microscope. (an indication of the destruction of lung tissue), but in gangrene, the elastic

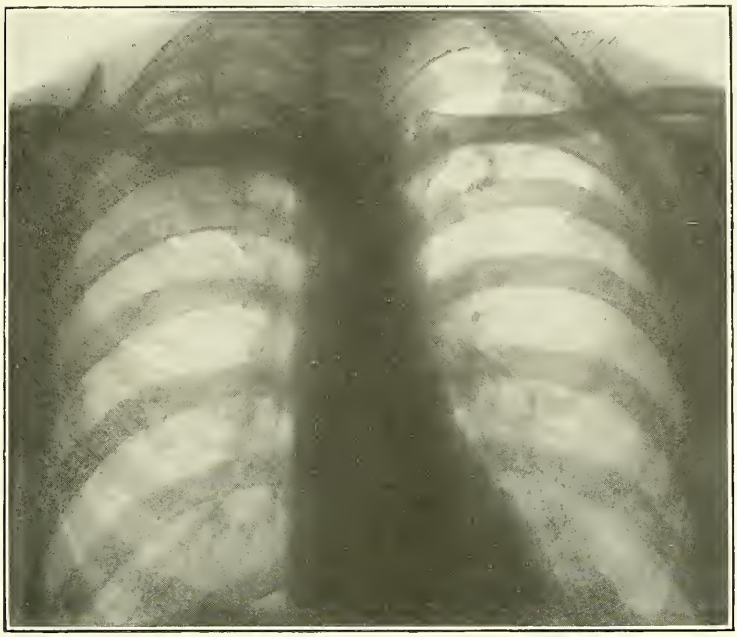


Fig. 39. Gangrene of the right upper lobe.

fibres are more difficult to find owing to the digestive action of the toxins. If allowed to stand in a cylindrical glass, the sputum separates into three layers; the upper consists of frothy mucus in which are shreds of tissue, the middle layer is watery and turbid, whilst the lower is viscid and contains the pus cells. This separation of the sputum into three layers is not pathognomonic of gangrene and abscess, it occurs also with the sputum of bronchiectasis and with the expectoration even of extra-pulmonary abscesses (empyema, subphrenic). The amount of sputum is no indication of the size of the cavity, as it is derived in part only from the suppurating focus and in part also from the inflamed and irritated mucous membrane of the bronchial tubes. The presence of the elastic fibres is of the greatest importance, as these are present only when there is breaking down of lung tissue. They serve as a means of differentiating gangrene and abscess from bronchiectasis (except those cases of bronchiectasis which are complicated by an abscess in the parenchyma); they assist also in distinguishing the intra- and the extra-pulmonary collections of pus, since these latter, even if they do rupture through the lung, do not necessarily cause extensive disintegration of that organ. Since the offensive odour of the sputum is due to the presence of saprophytic organisms associated with retention, it is present in some cases of bronchiectasis and extra-pulmonary abscess as well as in gangrene and abscess. In abscess and bronchiectasis, periodic recurrence of foul sputum associated with periodic imperfect drainage is common.

The signs of gangrene and chronic abscess vary greatly, according as to whether there is an acute and diffuse infiltration of the lung parenchyma without communication with a bronchus, or a well-defined abscess surrounded by consolidated lung with the pus escaping into the air passages. In the former case, a deficiency of movement on one side of the chest may be the sole localising sign, sometimes a tympanitic note can be obtained when percussing the chest over a deeply-seated focus. When the lesion is superficial the signs are those of pneumonic congestion, together with, possibly, those of pleural inflammation. The signs of a well-defined abscess depend on the extent of the surrounding consolidation, the depth of the lesion and the amount of pus in the cavity. As a chronic abscess may at one time be discharging freely and contain comparatively little pus, and at another time be distended owing to retention of the secretions, the clinical picture varies considerably at different times in the same patient.

RADIOLOGY.

In cases of *gangrene*, deficiency of movement of the affected side of the chest and of the diaphragm will be found. The gangrenous area shows as a dense shadow with a very irregular and in places often ill-defined

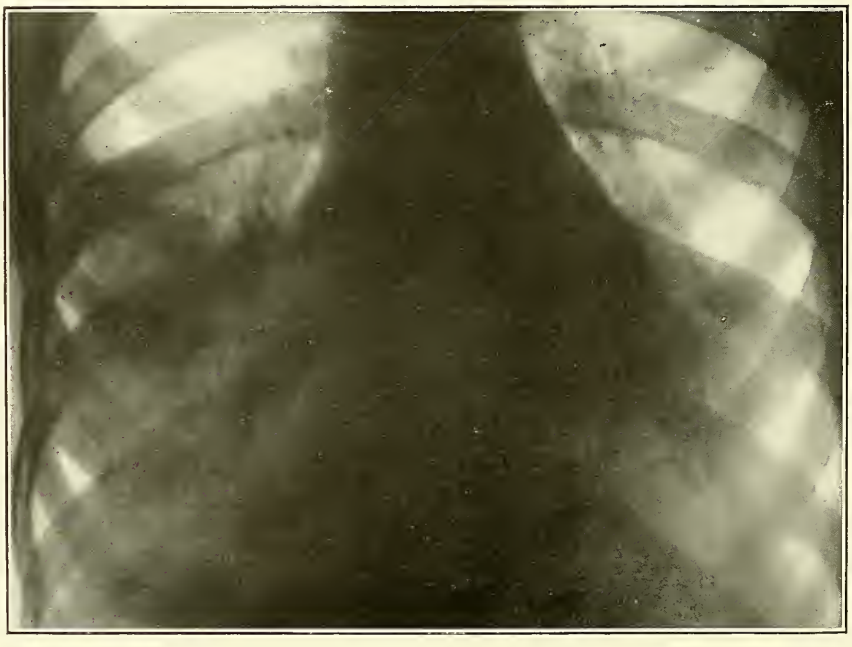


Fig. 40. Gangrene and broncho-pneumonia of middle lobe.

outline. The central zone is surrounded by a uniform but less dense shadow due to the pneumonic consolidation; this often involves the whole of the lobe, as can be seen in Fig. 40, in which the middle lobe is alone affected.* At times, the separation between the central and surrounding zones is not so distinct. When there are several gangrenous foci, these produce a series of darker shadows scattered about in the medium shadow of the consolidated lung.

A chronic abscess, when full, produces a dense circular shadow separated from the adjacent lung by a zone of less opacity. If empty, or partially so, the central opacity will be replaced by a dense ring with a

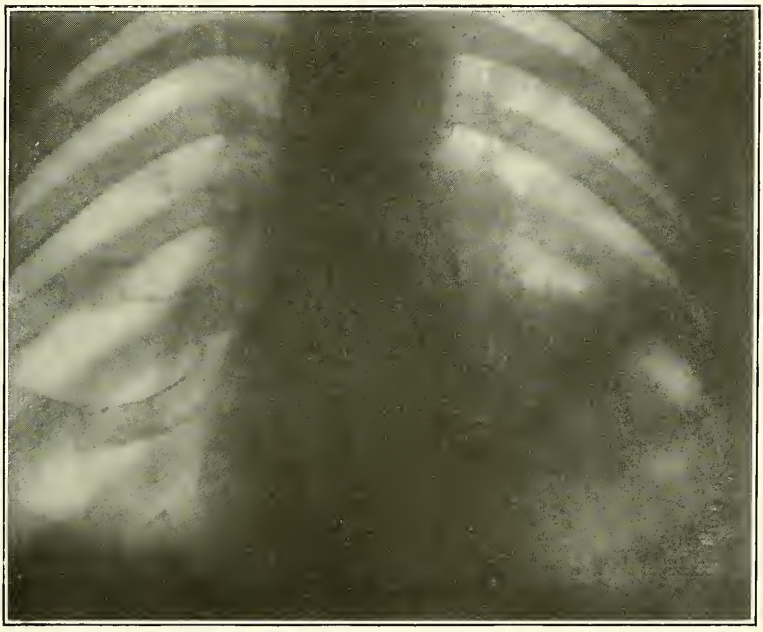


Fig. 41. Chronic abscess of left lower lobe. Surrounding pneumonic consolidation.

clear centre; or the lower segment of the circle will also be opaque and will have a horizontal upper border. When an acute is supervening on a chronic infection, the density of the shadow due to pneumonic consolidation, and possibly to the acute spread of the suppurative processes in the lung, is considerable, and it may be extremely difficult to recognise the limits of the abscess (see Fig. 41).

* Confirmed by autopsy twenty-four hours later. The gangrene in this case was secondary to a carcinoma of the œsophagus.

TREATMENT.

A very small percentage of cases of gangrene recover spontaneously. This result is dependent on the right combination of circumstances, such as the comparative mildness of the infection and the establishment of free and continuous drainage through the bronchus. The drainage of gangrene of the upper lobe is usually more efficient than is that of gangrene in the lower lobe, and the prognosis of the former group of cases is slightly better than of the latter. The mortality of gangrene treated by medical means is about 85 per cent., whereas surgical intervention reduces this to about a third. It is obvious, therefore, that the surgical is the correct treatment for all cases of gangrene which do not give early and definite evidence of spontaneous cure.

It is almost impossible for spontaneous cure to take place in cases of chronic abscess because the mechanical conditions are so unfavourable. Further, any treatment which does not include measures for overcoming these mechanical disabilities is incapable of accomplishing more than a temporary improvement in the patient's condition.

In all cases the exact localisation of the lesion before any actual treatment is undertaken is a matter of the first importance. This, even with radiography, is not always possible, and it is sometimes only by exploratory puncture that the exact depth and position of the focus can be determined. Exploratory puncture, unless every preparation has been made immediately to continue the operation, and it is possible therefore to leave the needle *in situ* when the point has entered the focus, is inadvisable, owing to the risks associated with it (*e.g.*, infection of the pleural cavity, or the sudden bursting of the abscess into a bronchus which is then flooded with the pus).

The methods of anæsthetising the patient have already been considered in Chapter II, p. 32.

The resistance of the patient to the shock of an anæsthetic and operation, specially when he is already exhausted by toxic absorption, can be greatly increased if he can assimilate large quantities of sugar during the preceding few days. The ingestion of sugar is of particular importance prior to operations on a suppurating lung lesion, because chloroform must be given in preference to ether and the toxic effect of this drug on the already partly exhausted liver cells may cause the death of the patient (delayed chloroform poisoning), unless special measures have been taken beforehand to combat the danger.

TREATMENT OF GANGRENE.

Once the diagnosis and the localisation of a gangrenous focus have been made, operation should not be delayed unless communication with a bronchus has already taken place and is followed by progressive and definite signs of improvement.

The operation consists (*a*) of the exact localisation of the focus by an exploring needle ; (*b*) the exclusion of the general pleural cavity from the area of operation (if this has not already been done by the formation of dense adhesions) ; (*c*) the incision into the gangrene and the establishment of free external drainage. These three steps should be done at one sitting.*

The exploratory puncture. The position of the patient on the table must be such as to ensure that there is no pressure on the sound side of the chest, whilst the affected area is accessible to the surgeon. A Record syringe of at least 20 c.c. capacity and two or three hollow needles, varying in length and diameter, are required. One needle should be at least 15 cm. long and 1 mm. in diameter. Puncture is made at first with the shorter needle attached to the syringe and then, if the gangrene is not found, with the longer. The punctures must be made through different spaces in varying directions. In some cases of gangrene, there is very little actual fluid within the affected area and to strike this with the needle is often impossible. If, however, the point of the needle has penetrated the gangrenous focus, the blood which is aspirated on withdrawing the plunger of the syringe will have an offensive odour and must be considered sufficient evidence. The needle, once it has struck the gangrene, is left *in situ* as a guide during the succeeding steps of the operation. Failure to locate the affected area is not uncommon. The operation should, nevertheless, be continued ; the difference in the technique in such cases is described later.

The suture of the pleural membranes. A vertical incision some four inches long is made ; it should cross the puncture hole of the needle and extend an equal distance above and below it, cutting through all the tissues down to the ribs. The margins of the incision are strongly retracted. The exposed portions, some 7 or 8 cm. in length, of three or four adjacent ribs are denuded of periosteum and excised.

It is never possible to determine before the operation whether the two pleural membranes are united by adhesions. In the majority of cases they are adherent, but in a proportion only of these cases again are the adhesions strong enough to prevent the leakage of pus into the adjacent parts of the pleural cavity, or to resist tearing during the manipulations on the lung. It is advisable, therefore, in all cases, to assume that no adhesions are present and to ensure the occlusion of the general pleural cavity from the area of operation by a series of carefully applied sutures. The parietal pleura and the visceral pleura and lung are very easily torn if any tension is made on the sutures, yet unless they are tied tight the membranes will not be brought into efficient apposition. The sutures should always include

* Some surgeons recommend that the operation be done in two stages—the exclusion of the pleural cavity at the first and the drainage of the gangrenous focus at the second. The delay of the forty-eight hours between these two may, however, be very serious to the patient, and the necessity of two operations, and possibly two anæsthetics, is detrimental for both physical and psychical reasons. By correct suturing of the pleural membranes, it is possible to avoid the danger of infecting the pleural cavity.

intercostal muscle or periosteum with the parietal pleura, and lung substance with the visceral pleura. If the stitches are inserted in the manner shown in Fig. 42, the tendency of these stitches to tear through the tissues will be minimal, whilst there will be close apposition of the pleural membranes. Each stitch must slightly overlap the preceding one and the line of sutures should enclose the area denuded of bone.

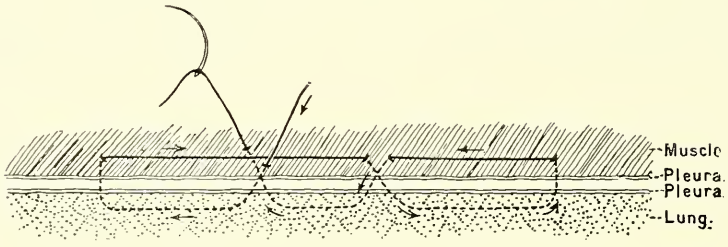


Fig. 42. Suture recommended by the Author for the approximation of the pleural membranes.

Incision into the lung. During the various procedures just detailed, the exploring needle has been left *in situ* and care should have been taken not to disturb it. An oblique incision is now made through the posterior layer of periosteum immediately above the needle. The incision, which extends to within 1 cm. of the pleural sutures on either side, divides periosteum, parietal pleura, visceral pleura, and penetrates into the substance of the lung, being so directed that it meets the side of the needle in the lung substance. The division of the lung tissue is then continued until the gangrenous focus is reached, all bleeding points being picked up and ligatured with catgut as soon as found, but no pause being made on account of general oozing.* The exploring needle is now withdrawn and the gloved finger introduced into the lung and the extent of the gangrene investigated. It must be remembered that no collection of pus is necessarily found, nor is there always a definite cavity ; the gangrenous area may consist of extremely friable lung tissue with perhaps a partially separated sphacelus surrounded by an irregular fissure which may be filled with a dirty-looking stinking fluid. Vessels, which may or may not be thrombosed, are occasionally felt as strands crossing the cavity. These should be ligatured as far as possible to prevent the occurrence of secondary hæmorrhage. If it is known that there are

* Some surgeons prefer to divide the lung with a thermo-cautery, as this checks the bleeding of the smaller vessels. The cautery, however, is not so satisfactory as the knife ; it is quite incapable of controlling the hæmorrhage from the larger vessels By charring the surface, it interferes with the recognition of the tissues which are being divided and may therefore penetrate a large vessel or a bronchus which could and should have been avoided.

secondary and neighbouring foci of gangrene, an attempt must be made to find these and to open them up into the main one. The wound is lightly plugged or a large drainage tube inserted, and the extremities of the cutaneous incision sutured.

There are many points which must be taken into consideration when determining which is the most satisfactory method of draining an acute gangrenous or a chronic abscess cavity. There can be no doubt that a drainage tube affords an easier outlet for the secretions from the cavity and allows of the escape of the sloughs as they separate. A drainage tube, provided that it is of large size, inhibits also, during the later stages of healing, the too rapid closure of the wound in the chest wall and the formation of a fistulous track. But, on the other hand, the rubber tube exercises no control on any oozing of blood that there may be from the walls of the cavity immediately after the operation, or on the hæmorrhage from a vessel which may give way at a later period, and, in fact, tends rather to encourage secondary hæmorrhage. If gauze is used as the means of conveying the infected secretions out of the cavity, it will help to check the bleeding, but it will prevent the expulsion of the separated sloughs until the plug is removed, and in the later stages it will be found more difficult to prevent the premature closure of the external opening. A gauze drain is, moreover, less readily changed and causes more pain than a drainage tube.

If there is a large bronchus or several smaller ones communicating freely with the cavity and there is a tube leading from this cavity to the outside, air would enter through the external opening during inspiration much more readily than through the glottis. There will be incomplete aeration of the blood and the patient may become intensely dyspnœic and cyanosed. During coughing, the air will escape easily through the wound in the chest wall, the explosive and expulsive force of the cough will be lost and the removal of the accumulating secretions will be greatly hindered. A part of the gauze drain, on the other hand, if such is used, may be drawn into one of the neighbouring bronchi and produce continuous and violent coughing owing to the irritation of the mucous lining.

As a general rule, it is advisable to drain the cavity at first by means of gauze, but to substitute for this as soon as possible a large drainage tube. The gauze must be moist and lightly inserted so as not to interfere with the escape of secretions. If, however, there is free hæmorrhage, the gauze must be packed tightly into all parts of the cavity.* The gauze drain should be changed every forty-eight hours, but it is advisable to leave it in for three or four days when there has been a severe hæmorrhage, and to give the patient a third of a grain of morphia before changing it.

* In cases of hæmorrhage, the gauze may be soaked in a warm solution of 20 grains of calcium chloride to the ounce of perchloride of mercury, 1 in 5,000.

In some cases, on opening the gangrene, or subsequent to the operation, there is a severe secondary hæmorrhage, the blood pouring out of the wound and through the bronchus out of the mouth. The wound must be immediately packed with a long strip of narrow gauze, care being taken that the packing extends to the end and fills the cavity. A hypodermic injection of morphia is also given. If the gangrene is offensive, it will be necessary to change the gauze in forty-eight hours. Morphia should again be given a quarter of an hour before the dressing, and a fresh plug be prepared ready to insert the moment the soiled one is removed. These precautions are necessary at each subsequent dressing until the plug can be changed without bleeding.

The procedures detailed above are those necessary when opening a gangrenous focus which has been located by the exploring needle. The technique is slightly different when gangrene of a lobe is known to exist, but the exact situation of it cannot be found by puncture. The incision, in such cases, is made over that part where the physical signs suggest that the lesion is nearest to the surface. The removal of the ribs, the suture of the pleural membranes and the preliminary incision through the periosteum and pleura into the substance of the lung are the same as described above. It is always advisable again to explore with a hollow needle as soon as the visceral pleura has been incised, the needle being introduced through the exposed lung tissue. The course to be adopted if there is once more failure to locate the gangrene depends upon the general condition of the patient.

In a case in which the patient is seriously ill owing to the toxic character of the lesion, it is advisable to continue the incision into the deeper parts of the lung substance, and to explore the adjacent surfaces of the parenchyma with the finger. An area of increased or diminished resistance should arouse suspicion of the proximity of the lesion ; the former is due to consolidation and the latter to the friable lung. Care must be taken that the resistance caused by a bronchus and the adjacent vessels is not mistaken for consolidation. Should the focus even now remain undiscovered, the wound must be lightly packed with gauze in the hope that, if the gangrene spreads, it will open up into the wound.* In such a case, it will be possible later to enlarge the communication with the finger ; there is no necessity to give the patient an anæsthetic for this last procedure.

When the general condition of the patient is good, it is preferable, having made the incision into the surface of the lung and having failed by repeated puncture to find the gangrene, to postpone the rest of the operation until further investigations in order to locate the lesion have been made. If it is possible again to radiograph the chest, great assistance may be obtained by inserting at the time of the operation a blunt probe, 5 inches long, through

* Some surgeons recommend that a second incision should be made at right angles to the first, so as to open up a larger area of lung tissue.

the wound into the lung parenchyma.* The relation of the shadow of this probe to the shadow of the gangrene will help materially to ensure success at the next operation, which should be done within forty-eight hours.

After treatment. In a straightforward case, the character of the discharge will change considerably in the first few days after drainage has been established. If at first it is watery, turbid and offensive, containing fragments of lung tissue, it will become purulent and the odour will diminish as soon as the necrosed portions of lung have sloughed away. If, when the gangrene is opened, there is a cavity containing pus, the discharge will gradually become muco-purulent. With the cessation of the offensive discharge and the separation of the sloughs, granulations will form and the cavity will contract down on to the drainage tube. The drainage tube used should be large, with the inner end cut obliquely. As the discharge alters in character and decreases in amount, the tube may be gradually shortened. It is never advisable to diminish the calibre of the tube unless compelled to by the constriction of the surrounding walls. If the gangrene has been operated on before there has been time for delimiting walls to be formed, a later plastic operation is very rarely necessary.

Cases of gangrene which have ruptured into the pleural cavity giving rise to a secondary empyema are mostly hopeless, but as it is occasionally possible to save life, they should always be treated by operation. At first, only a large opening into the lower part of the pleural cavity should be made; this will allow of a free drainage of the empyema, and, as the gangrene has already opened into the pleura, it may indirectly suffice for the drainage of the pulmonary lesion, particularly when the affected area is small and superficial. For this reason and in consideration of the fact that the patient could not tolerate a bigger operation at the time for the treatment of the gangrenous foci itself, no more active intervention is justifiable at this stage. After three or four days it will be possible to determine whether direct intervention for the lung lesion is necessary, the indications being a continuation of the symptoms of gangrene, the cough, the fœtid expectoration and the swinging temperature, together with a continuance of an offensive discharge from the empyema cavity. One difficulty of the operation is that the position of the gangrenous focus, even if it had been located before rupture into the pleura, can no longer be ascertained owing to the collapse of the lung. If further treatment is decided on, the original empyema wound must be considerably enlarged (when it is known or suspected that the gangrene is in the lower lobe) by prolonging the incision along the rib and removing another 5 to 8 cm. of it. Through this opening the lung can be examined by reflected light, or the hand can be inserted and the organ palpated. When the point of rupture is found, this must be enlarged so that the gangrene can drain with the utmost freedom *via* the pleural cavity.

* Thread should be tied to the eye of the probe and fixed by strapping to the chest wall.

Re-expansion of the lung, whether the gangrene has cleared up spontaneously after drainage of the empyema or secondarily after enlarging the opening, is practically never complete and a chronic discharging empyema results. This, when the patient has completely convalesced from the gangrene, must be treated in the manner described on pp. 77 to 80.

It has previously been emphasised that empyema cavities should never be irrigated with fluids. Irrigation, however, with ozone, allowed to enter without any force and with a large aperture for its escape, may well be tried in cases where there is persistence of an offensive odour in the discharge. Autogenous vaccines also are sometimes of assistance in cases of gangrene, especially when there is a predominance of one type of organism. Vaccines, however, must be used as an adjuvant only to free incision and drainage.

TREATMENT OF CHRONIC ABSCESS.

The primary object of the operative treatment of both gangrene and chronic abscess is to make a free opening for the escape of the discharge and of the products of disintegration, and so to diminish at first and then arrest the processes of absorption of the toxins, at the same time reducing the cough and expectoration. This treatment by free incision and drainage is sufficient to effect a cure of an acute gangrenous focus, but in the treatment of chronic abscess it is inadequate. The fundamental difference between the two states is that fibrotic changes are practically non-existent in gangrene; they are well marked in chronic abscess. In the former there is no well-defined cavity and as the devitalised tissues slough and escape through the wound, comparatively little readjustment of the surrounding tissues is necessary to enable the walls to come into apposition, and to coalesce when the raw areas are covered by granulation tissue. The loss in volume is easily made up for by slight compensatory emphysema of the healthy lung. In chronic abscess, there is often a big cavity and it is bounded by densely fibrotic walls which, even when the cavity is empty, are prevented from coming together by the rigidity of the fibrous tissue. It is evident therefore that simple drainage must be supplemented by some procedure which will overcome the mechanical disabilities and enable the approximation of the walls to take place. The most satisfactory means for accomplishing this is wide decostalisation of that region of the chest wall which is superficial to the abscess cavity. This essential part of the operation should be done at the time of opening the abscess.

It has already been stated that the infection in a chronic abscess may suddenly increase in virulence and lead to ulceration of the walls of the cavity, and to gangrene. On the other hand, the course taken by the disease may be influenced by variations in the intensity of the sepsis due to re-infection of the cavity or to retention of the pus, causing fever and other

signs of toxic absorption, but not of such an acute nature as to suggest the onset of a gangrenous process. In the former case immediate operation is necessary as the only means of preventing the spread of the disease ; but in the latter type, operation should be deferred until the toxic condition has been reduced to a minimum. To this end expectorants should be given and the patient inverted* morning and evening in order, as far as possible, periodically to empty the cavity. An autogenous vaccine may also be given, but only when the cavity is discharging freely, that is to say, not during a period when the pus is pent up.

The pus in an abscess cavity is normally exposed to an equal pressure on all sides of it. When the patient coughs, the pressure on the walls of the cavity, at the moment that the glottis is opened, is greater than in the bronchi and the pus is therefore expelled into the bronchus and along the trachea. The inspiratory act is always gentle, the expiratory only is forcible and is persisted with until the mucus or pus is dislodged from the bronchi. When the patient is under the influence of an anæsthetic and the cough reflex is lost, the escape of pus into the bronchial tubes fails to elicit the reflex expulsive mechanism, whilst the inspiratory act is almost as energetic as the expiratory ; it may (during sighing or gasping respiration) be sufficiently powerful to draw the pus down into the bronchi leading to the healthy as well as to the diseased part of the lung. The effect of this aspiration is to produce instantaneous and complete blocking of a considerable number of the smaller air passages and possibly immediate death from asphyxia. This constitutes one of the gravest dangers of operations on pulmonary abscesses. The pus may be forced out of the cavity as a result of the manipulations on the wall of the chest or of the cavity itself ; but the danger is greatest at the moment of opening the abscess. The air in the bronchi is at a negative pressure, whilst that outside the cavity (when the incision has extended down to it) is at that of the atmosphere. As soon, therefore, as the wall of this cavity is incised, the pus is both sucked and driven into the bronchus. The great importance of the intratracheal method of administering the anæsthetic as a prophylactic measure against this danger has already been discussed in Chapter II.

The position of the patient on the operating table, the localisation of the abscess by puncture, the removal of the ribs, the suture of the pleural membranes and the incision into the abscess cavity are done in precisely the same way as has already been detailed in the description of the operation for gangrene. There is this variation, however—whereas in gangrene the number and length of ribs removed is comparatively small, in cases of abscess the rib removal should be as extensive as possible ; at the very least the whole of the bony parts overlying the cavity and some 3 or 4 cm. of surrounding lung tissue must be cut away. When the incision through the wall of the

* The patient should hang over the edge of the bed, resting with his hands on the floor, and the pelvis and lower limbs on the bed.

abscess cavity has been made, it is advisable to bring the margins of the incision through the lung up to the wound in the chest wall and to suture them there with strong catgut mattress sutures. This procedure is not only an additional aid against the infection of the pleural cavity, but assists also in maintaining a free opening into the interior of the cavity and allows of exploration without the danger of separating the lung from the parietal pleura. If strands of tissue are felt crossing the abscess cavity, these must be ligatured, as they almost certainly contain vessels, and if left untreated they will, unless thrombosed, either dilate and form small aneurisms and eventually rupture, or, as the result of ulceration of the coats, give rise to secondary hæmorrhage.

The relative value of moist gauze or rubber tubing for the drainage of the cavity has already been discussed in connection with gangrene. In the case of a chronic abscess, the need of affording a free exit for sloughs is not so great, whilst there is a considerably greater risk of inefficient aeration of the lungs and of the loss of the explosive force required to expel secretions, owing to the freedom of the opening usually present between the abscess cavity and one or more bronchi. The gauze drain,* even when supplemented by plentiful dressings on the outside,† is sometimes not sufficient to prevent the entrance and escape of air through the external wound. If symptoms of distress develop from this cause, they can be abolished by laying over the wound a square of sterilised oil-silk, three sides of which are fixed to the chest by strapping, while from under the fourth, the gauze drain is brought out so as to carry the secretions into the dressings.

It is recommended by some surgeons that in all cases of chronic abscess the operation should be done in two stages—the first consisting of those steps down to and including the suture of the pleural membranes, the second, of the division of the pleura and the incision into the abscess. When the cavity drains freely into the bronchus and is found by puncture, there is no particular object in delaying the actual opening of the abscess, even when the pleural membranes are non-adherent, provided that the surgeon is satisfied that his sutures have effected close approximation of the parietal and visceral membranes. In those cases, however, in which drainage is imperfect, and in which therefore the abscess cavity tends to fill up unless the patient is regularly inverted, there are certain advantages in the two-stage operation, but there are also some disadvantages. The first stage is done under a general anæsthetic with the abscess cavity emptied as far as possible by inversion of the patient during the preceding few days and immediately before the operation; whilst the second stage is done a few days later,

* If the gauze is packed into the wound sufficiently tightly to prevent the free passage of air, it will act as a plug damming back the secretions and not as a drain.

† Owing to the movements of the chest wall the dressings after a short time always become loosened and cease to form a firm obstructing pad.

the pus having been allowed to accumulate. On this occasion a large dose of morphia only is given. The advantages claimed for this method are that the dangers, whilst giving the general anæsthetic, are reduced to a minimum, and that the distended abscess cavity is, at the second stage, much more easily found. Moreover, during the period between the first and second stages there is a strong probability, though not a certainty, that firm union will have taken place between the two pleural membranes. The introduction of intratracheal anæsthesia has in a great measure abolished the danger of aspiration of the pus into other parts of the lung and it has become safer to open the abscess, even when full, with the use of the insufflation method of anæsthesia rather than under morphia. The morphia, though leaving the patient his cough reflex, does not thereby necessarily avert the danger of aspiration of a large quantity of pus at the moment of incising the abscess. With intratracheal insufflation, the positive pressure inside the trachea drives the pus out through the external opening and abolishes all risk of sudden flooding of the bronchi and bronchioles. A second disadvantage of the two-stage method is that the first operation must be done with the abscess cavity as empty as possible (when an ordinary anæsthetic is given) and this necessarily increases the difficulty of finding the abscess by exploratory puncture.

It follows from what has been said above, that when the abscess cavity is one in which the drainage is imperfect, but can be controlled by the position of the patient, it is advisable always, as a preliminary to operation, to keep the cavity as empty as possible by repeated inversion, so as to improve the general state of the patient. If intratracheal insufflation anæsthesia* is available, the cavity may be allowed to fill up immediately before the operation, all the steps of which, including the incision into the cavity, can then be done in one stage. If, on the other hand, no such special apparatus is at hand, it is safer to do the operation in two stages as detailed above, giving the patient a general anæsthetic for the first and morphia only for the second.

It occasionally happens that a chronic abscess, the symptoms of which are indisputable, cannot be accurately localised because the signs are not sufficiently definite, and because either X-rays are not available or the radiogram when taken does not give the required information owing to the extent of the surrounding pneumonic consolidation. Such a case should always be regarded with considerable suspicion, as it is possibly one in which the abscess is secondary to a bronchiectasis, or the abscess is primary and is complicated by a bronchiectatic condition ; for either of which alternatives incision and drainage is not the correct treatment. When, however, the

* It must be understood that in all cases in which the intratracheal insufflation method of giving the anæsthetic is recommended, whether because of the dangers of aspiration of pus or of those of an uncompensated pneumothorax, this method is not advocated in preference to the negative pressure chamber, which undoubtedly is the more satisfactory, but it is mentioned as being the form of apparatus in most constant use.

evidence indicates that there is a cavity which must be treated by the open method, and repeated puncture fails to disclose the seat of the pus, it is advisable to make an incision through the chest wall sufficiently large to admit the hand into the pleural cavity. The lung can then be palpated and, provided that the diagnosis is correct, the abscess cavity can be easily recognised. The surface of the lung nearest to the abscess must, if possible, be brought up to the opening in the chest wall and secured to its margins ; but if this is impracticable, it should be approximated to that part of the chest wall with which it is normally in contact, and secured there by two stout catgut stitches passed through the adjoining intercostal spaces.* The exploratory incision is then securely closed and the air in the pleural cavity replaced by oxygen at a negative pressure. At the end of a week the lung should be completely re-expanded, and the exact situation of the abscess being known, the operation can now be undertaken in the manner already described in the preceding pages.

The object of the operative treatment of chronic abscesses is to obtain complete obliteration of the cavity, and for this it is essential that the walls should be approximated. This cannot be effected solely by the displacement of the mediastinum and diaphragm towards the affected part and by the expansion of the surrounding healthy parts of the lung. Closure of an abscess cavity, especially when that cavity communicates with a bronchus, is always hard to obtain and every means must be taken to encourage the process. It has already been pointed out that a free collapse of the chest wall is a necessary adjuvant, but it may be that the condition of the patient at the time of the operation does not justify further interference than is necessary for making an opening into the lung. If nothing more is done a chronic sinus or a bronchial fistula will result. Such an operation, therefore, should always be considered from the first as incomplete, and the more extensive rib resection must be carried out as soon as the patient has sufficiently recovered. It is most unwise to delay the completion " on the chance " that the cavity will become obliterated.

The continuance of an offensive odour in the pus, or the reappearance of this symptom after it has already subsided, is an indication either that the external drainage is inadequate on account of the size and position of the opening and that there is consequently retention of the secretions, or that there is a spread of the processes of infection in the lung substance.

The lines of treatment which have already been laid down are those suitable for cases of single abscesses without associated extensive dilatation of the bronchi, and are not applicable to multiple chronic abscesses or to single abscesses complicated by bronchiectasis. The most efficacious method

* As it is not advisable that these stitches should pass through the skin, owing to the risk of inflecting the pleural cavity, a small incision should be made through the cutaneous tissues before introducing the suture.

of dealing with these varieties of chronic suppuration is by the operation of rib mobilisation, which produces a very extensive collapse of the chest wall and consequently of the lung also. This operation is described in detail in Chapter IX.

POST-OPERATIVE COMPLICATIONS.

There is always the danger of a spread of infection when treating acute and chronic suppurative processes in the lung. The possibility of septic pneumonia, pleurisy, empyema, cellulitis or gangrene of the chest wall and pericarditis must be thought of when there is an exacerbation of symptoms while the wound itself is giving no cause for anxiety.

In all operations on any chronic septic foci in the thorax, there is the risk of a cerebral abscess appearing as a complication during the ensuing ten days; this complication is referred to more fully in the chapter on bronchiectasis.

Chapter VIII.

PULMONARY TUBERCULOSIS.

Pulmonary tuberculosis, by the time it is recognised or the patient presents himself for treatment, has usually passed through that comparatively brief period during which the tubercular infection exists as a simple condition. The clinical picture of acute miliary tuberculosis is rare. When the disease breaks out as an irresistible invasion of the lung, there are soon added the signs of extensive pneumonic consolidation or of scattered areas of broncho-pneumonia in the lobe or lung. In the chronic type also, with which we are mainly concerned, once the organisms have established themselves, it is not so very long before the secondary changes consequent on the tuberculosis begin to assert themselves. The chief of these is fibrosis, and this in turn is responsible for the progressive and ultimately extensive intrathoracic mechanical disabilities and eventually for bronchial distortion and dilatation. Sooner or later, secondary infection by pyogenic and saprophytic organisms supervenes on the primary lesion. This secondary infection has its own group of complications, the chief of which is hæmorrhage.

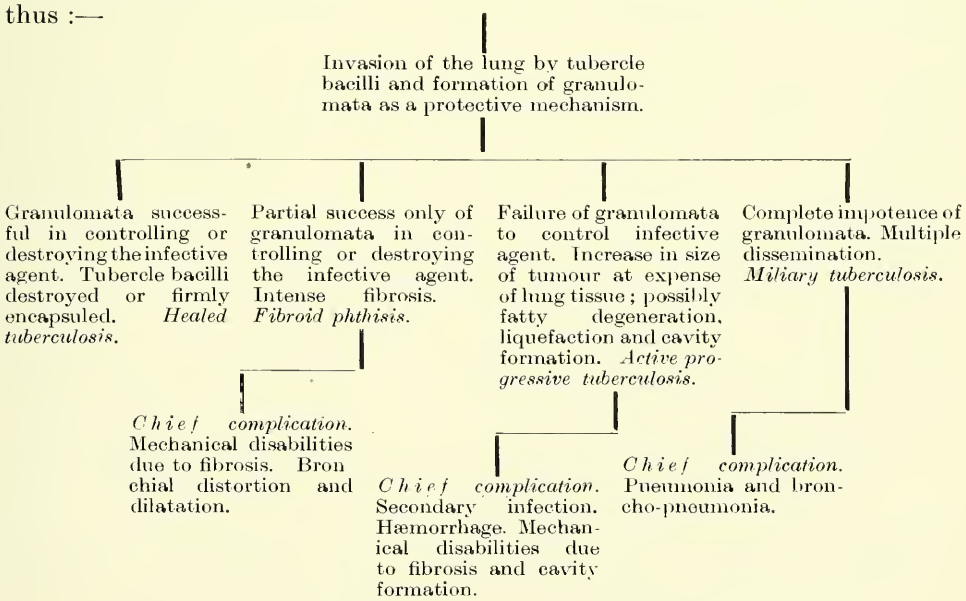
It is obvious, therefore, that the clinical pictures which can be seen in the course of the disease may be many and extremely varied, and this not only in different patients, but in the same patient at different stages in the disease. It is of comparatively little value in cases in which the disease is indubitably present to label the condition from which the patient is suffering as pulmonary tuberculosis, or without further definition to attempt to treat the patient on that diagnosis. It is absolutely essential, if good results are to be obtained, that the clinical picture should be translated into terms indicative of the pathological lesion, that is to say, into terms which denote the activity of the primary lesion and the extent of the secondary changes due to it—the fibrosis, the mechanical disabilities, cavity formation and distortion of the bronchi, the presence also of any superadded infection and the influence which it is exerting in the course of the disease. This it is quite impossible to do without a precise knowledge of the pathology of the disease and of its influence on the normal physiological and anatomical conditions in the chest.

Tuberculosis manifests itself pathologically as a granuloma. A granuloma is a tumour composed of granulation tissue which is formed by the reaction of the normal tissues round the organisms in response to and as a protection from the continuous mild irritation they exercise. The function of this granuloma is to enclose and to destroy the bacilli. The granulation tissue when it has accomplished its work becomes converted into fibrous tissue. Under suitable conditions, therefore, the granuloma must be

regarded as a benign tumour which appears in response to the call for protection and, if successful, disappears, leaving behind a small scar as the sole indication of its beneficent intervention. But if the granuloma fails to control or to destroy the infective agent, then a change in its effect takes place, and it comes to exercise a destructive action on the surrounding tissues resembling that of a malignant tumour. When the general or local conditions are so unsatisfactory that the granuloma is unable to overcome the invading bacilli, and they multiply freely, the formation of granulation tissue increases rapidly at the expense of the surrounding lung parenchyma which it destroys.* Just as a rodent ulcer can heal in one part, the malignant cells dying and being replaced by a fibrous scar while it continues its destructive spread in other regions, so may a granuloma simultaneously cause fibrosis and destruction.

The blood supply of a granuloma is derived essentially from capillaries. Where the tumour through failure to control the infection increases indefinitely in size, a point must be reached when the blood supply ceases to be adequate. The result of this is fatty degeneration of the central parts of the tumour; this is followed by caseation and liquefaction. When the stage of liquefaction has been reached, the containing walls give way into a bronchus and the space that is left behind forms a cavity in the lung. Other foci which have reached this degree of disintegration may also open into the same cavity.

The various pathological processes may be represented diagrammatically thus :—



*Its power of destruction is not equal on all structures and dense fibrous tissue, such as the pleura, will resist its onslaught for a considerable time. The granuloma of tubercle is very similar to the granuloma of syphilis. In those cases where fibrosis is considerable (fibroid phthisis), the change resembles closely that found in a syphilitic gumma. The selective action shown in the destructive action on the tissues is one of the few features absolutely characteristic of tubercle.

It is obvious that the future of a person whose lungs have become invaded by tubercle bacilli depends primarily on the degree of success or failure of the granuloma in controlling the infective processes, and it is necessary, therefore, to consider by what conditions the functioning of the granuloma is influenced. The essential factors are three in number. They are :—

(1) The general resistance of the patient to tuberculosis.

(2) The dose of the infection measured either by quantity or by virulence.

(3) The local conditions to which the granuloma is exposed.

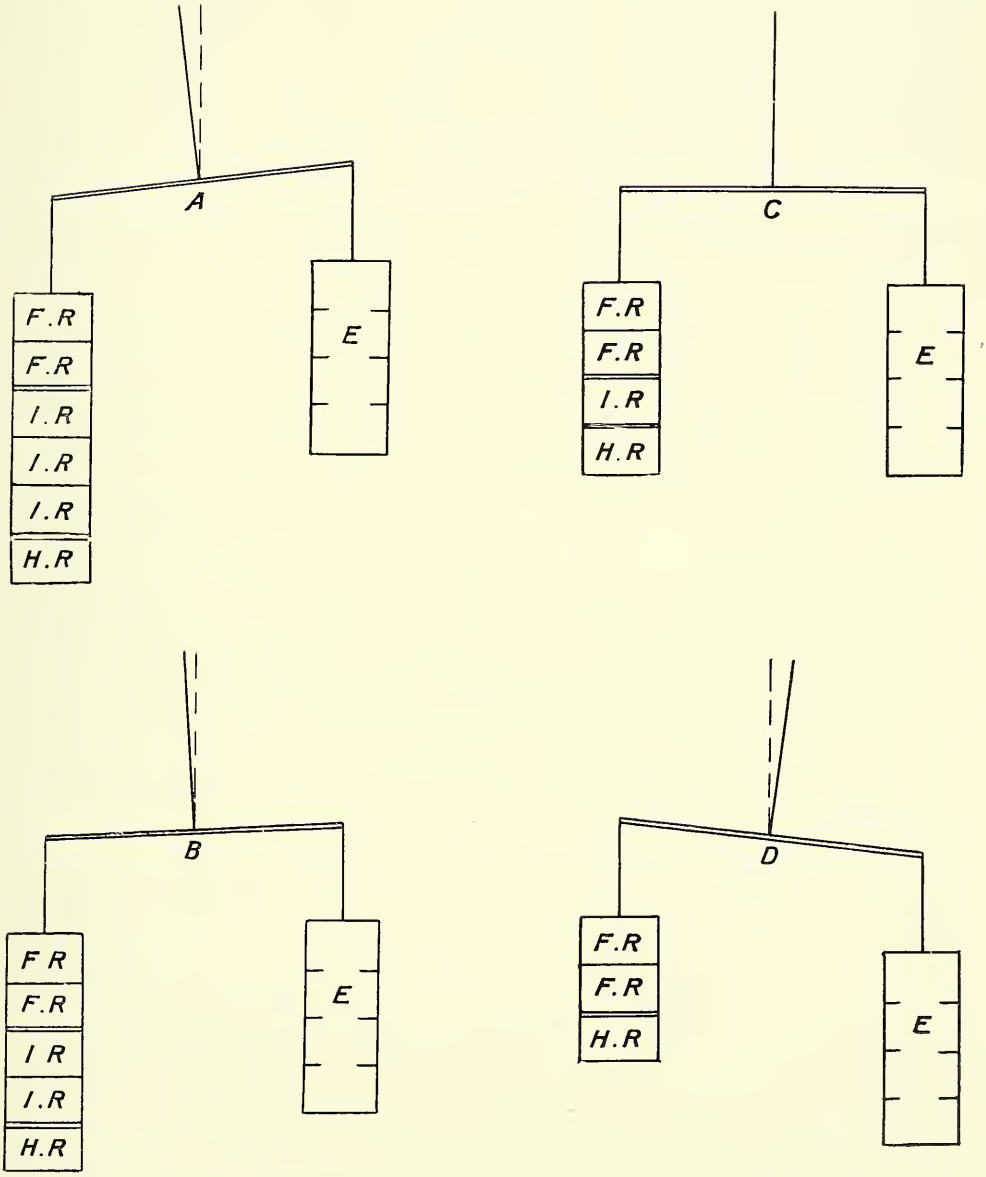
The development of pulmonary tuberculosis and the spread of the disease depends primarily on the degree of resistance of the body to the infection. Some people enjoy complete immunity, in others there is such an absence of resistance that if exposed to an adequate dose of tubercle bacilli, they succumb rapidly to a fulminating tuberculosis. Between these two extremes, with which we are not now concerned, there is a gradation of resistance so fine and so variable, owing to the influence of other factors, as to form an apparently continuous series. It must be obvious, however, that there is some intermediate point at which, we may suppose, equilibrium to exist under normal conditions. If the equilibrium is disturbed by external influences, such as insufficient food, overwork, bad hygiene, an infection of the nature of influenza or local trauma of the lung, the resistance of the body becomes decreased and the balance is disturbed in an adverse direction. Exposure at this stage to a dose of tubercle bacilli would lead probably to the development of pulmonary tuberculosis, and to the spread of the disease as long as the equilibrium is lost. This illustration can be extended and shown diagrammatically as on the page opposite.

The resistance of any given individual to tuberculosis is conceived of as being made up of three factors :—

(1) The fixed resistance of the species common to all and beyond the reach of individual variations.

(2) The individual resistance—the inherited resistance characteristic of the given individual, varying from person to person, but constant in the same individual.

(3) The health resistance—that factor in the power of resisting tuberculosis that depends on the general health of the individual. When (2) is well marked, the individual will not get tuberculosis whether his health is good or bad ; when (2) is ill-developed, the individual is dependent on the state of his general health for protection against tuberculosis.



F.R. = Fixed resistance.
H.R. = Health resistance.
I.R. = Individual resistance.
E. = Average exposure.

N.B.—The resistance is represented in terms of weight.

In A, resistance to the disease is strong and able to resist all ordinary exposures to infection. In D, the resistance is much lower than normal and exposure to the average risks of infection will result in acute tuberculosis. C shows a state of unstable equilibrium. The resistance is not more than just adequate when the general health is good. When the general condition is reduced, average risks of exposure will result in tuberculosis of a progressive and more or less intractable type. In B, the state of the individual may be regarded as intermediate between A and C. In sound health the patient resists tuberculosis well. Should he become infected during a period of depressed health, he will respond favourably to sanatorium treatment. Such a patient is of the not very numerous class that, having developed manifest tuberculosis, is cured in a sanatorium.

It must be very clearly understood that there is actually no such limitation of the disease or of the power of resistance of individuals into groups, but that in reality every degree of resistance between the extreme of A and the extreme of D is to be met with, and that the four groups illustrated above represent nothing more than four widely separated degrees picked out for the sake of clearness of description.

This diagrammatic method of representing the circumstances which influence the onset and the course of the disease can be utilised to illustrate certain broad general principles derived from clinical experience of treatment. Thus treatment limited to the improvement of the general state of health is powerless to influence cases of the acute type such as occur in group D. It may, on the other hand, be all sufficing for those which fall in group B, especially if the treatment is started in the earlier stages. The effect of such treatment on the class of case which belongs to group C is to produce a very considerable improvement in the general health of the patient but without any corresponding improvement in the disease, or arrest of even the active processes.

The local conditions to which the granuloma may be exposed are best considered in connection with tuberculosis of some other part of the body, *e.g.*, joints, or tendon sheaths, the effect of direct treatment on which we are more conversant with. It is a well-known fact that the complete immobilisation of the affected region is not only the most important part of the treatment, but is frequently quite sufficient in itself to produce a cure. No benefit can be obtained, nor is it expected, by simply improving the general health of the patient without any concomitant attempt to abolish local movement. This does not mean that improvement to the general health is valueless and therefore unnecessary, but unless combined with local immobilisation it is incapable of overcoming the adverse influences of constant movement. It is obvious from this that one of the essentials for the successful evolution of the physiological function of the granuloma is rest, and there is no reason to suppose that a granuloma of the lung differs in this respect from a granuloma in any other part of the body. What can

be regarded as surprising is that pulmonary tuberculosis may undoubtedly heal without any other treatment than that calculated to produce the increase of resistance which can be contributed by an improvement of the general health. The credit for this belongs to the lung parenchyma, the great vascularity of which is most favourable to the granuloma and enables it to accomplish its object under conditions which would be impossible elsewhere. It must be remembered that the lung is in constant motion and that this is increased both in rate and extent by exercise. When the patient is suffering from tuberculosis, the movements occasioned by the cough and the attempts to get rid of the sputum necessarily produce frequent and energetic disturbance of the lung. Rest in bed diminishes the rate of inspiration and expiration, and it may reduce but cannot abolish the cough. It cannot, therefore, be regarded as in any way comparable with the immobilisation insisted on by the surgeon in the treatment of a tuberculous joint.

So far the changes directly associated with the granuloma have been dealt with, and it is necessary now to consider the mechanical disabilities which are the result of the antagonism between the fibrosis produced by the granuloma during its progress and the normal intrathoracic physiological and anatomical conditions.

The constant tendency of fibrous tissue is to contract ; the more rapidly it is produced and the more intense the irritation responsible for the production of it, the greater is the contractile tendency of this tissue. When the irritation responsible for the fibrosis has ceased, and the fibrous tissue is completely formed, it does not tend to go on contracting indefinitely, and will, indeed, even yield to constant gentle traction.

The contraction of the fibrous tissue in the lung leads to a diminution in the volume of the organ. This diminution must necessarily be compensated for by changes in the surrounding walls, by changes in the lung itself and by increase in the intrapleural negative pressure. This last factor, however, plays a comparatively small part, as even in cases of very long and marked fibrosis it is rare to find an increase in the negative pressure equal to more than 2 mm. of mercury.

The changes in the surrounding wall, while considerable, cannot extend beyond a certain stage, which stage is, however, variable in different individuals. The changes consist in displacement of the various walls inwards to the affected lung. The extent of the displacement is dependent, so far as the chest wall is concerned, on the flexibility of the cartilages, which is, of course, greatest in young people ; so far as the mediastinum is concerned, on the rigidity of the partition, which is less dependent on age than on individual peculiarity ; and so far as the diaphragm is concerned, on the presence of adhesions between the lung and the muscle. The diaphragm responds but little to an indirect pull, that is to say, to a pull which is exerted through the medium of the negative pressure only, as the under surface is

not exposed to the full atmospheric pressure ; but considerable distortion may be produced by direct pull through adhesions.

Some compensation for the diminution in volume is also effected by emphysema of the unaffected parts of the implicated lung. The displacement of the mediastinum is associated with enlargement of the opposite pleural cavity, and this in turn must be compensated for by emphysema of that lung. When both lungs are infected by tuberculosis and involved by fibrosis, there is some displacement of the mediastinum to the side primarily involved by the disease, since the fibrous tissue formation started first in this lung and the diminution of the organ is consequently greater. Necessarily, however, the degree to which the mediastinum can compensate for the diminution in the volume of the lung is less.

As has already been stated, the extent to which the surrounding walls and emphysema of the healthy parts of the lung can compensate for the diminution in the volume due to the contraction of the fibrous tissue is limited, and when that limit has been reached, the tuberculosis can certainly be regarded as having passed beyond the early stages of the disease. An " early case " of pulmonary tuberculosis can in fact be defined as one in which the shrinkage of the lung necessary for cicatrisation can be compensated *fully* by the yielding of the surrounding parts.

The influence of the fibrosis is not, however, in progressive and late cases, limited to those changes which have just been described. The surrounding walls offer a considerable resistance to the pull of the fibrous tissue. If it were not so the whole effect of the pull would be exerted at what may be described as the free end of the fibrous tissue at the periphery of the lung, and the circumference of the lung would be drawn down to the fixed end at the hilum. But owing to the external resistance, the traction of the fibrous tissue is central as well as peripheral, and as a result of this there is a drag on the walls of the bronchi which become progressively distorted and dilated. Bronchial dilatation (secondary bronchiectasis) is also produced by the fibrous tissue, when it is massive, exerting its influence by constricting the bronchi at various points. The effect of this is to produce dilatation of the tubes in their course distal to the constriction.

Abnormal cavitation occurs as a direct sequence of the pathological changes commonly found in pulmonary tuberculosis—cavity formation in the parenchyma due to caseation of the granuloma* and dilatation of the bronchi due to constriction or direct pull on the walls. The inevitable result of cavitation is secondary infection by pyogenic and saprophytic organisms, unless the drainage is so free that there is never any retention of secretion. The blood vessels and the bronchi are very resistant to invasion by the granuloma and consequently these structures are frequently to be found traversing a cavity in the parenchyma. The vessels may be more or

* Such a cavity is not necessarily sufficiently large to produce clinical signs so characteristic that the condition can be recognised.

less normal ; they may be thrombosed, or there may be aneurismal dilatation of the walls owing to the lack of support. Such an aneurism may rupture during a fit of coughing and cause a profuse hæmoptysis. Hæmorrhage may result also from ulceration of the lung parenchyma as a result of the irritation caused by secondary infection by pyogenic organisms.

Hæmoptysis does not occur only during that stage of the disease when secondary infection is indubitably present. It may be the first recognisable symptom which calls attention to the pulmonary lesion, appearing at a time when the existence of a pyogenic infection is extremely difficult or even impossible of demonstration. It is conceivable that the hæmoptysis may at this comparatively early period be due to the congestion of the mucous membrane caused by the irritation of the underlying granuloma. It is equally possible that it is associated with the rupture of a granuloma which has undergone degeneration, and it is difficult to suppose that, once the rupture has occurred, the cavity which results can long remain free from secondary infection.

It is probably erroneous to suppose that the irritation occasioned by one or two isolated granulomata, or that the changes which are associated with the process of caseation and liquefaction in them or with their rupture must necessarily cause symptoms pronounced in character. So many people suffer from slight chronic irritation of the air passages or transient infections, that it requires some symptom as startling as hæmoptysis, or the continued persistence of cough and expectoration with a decline in the general health, to raise suspicion or to lead to an investigation.

There is one further fact in connection with cavities which must be mentioned—beyond the walls there is often an encircling zone of pneumonic consolidation. The presence and the extent of this zone are an indication of the virulence of the secondary infection. A broncho-pneumonia may occur as a result of extensive invasion of a bronchiole by granulomata or possibly of the spread of infection by inhalation.

When either the primary or secondary infective processes approach the surface of the lung they tend to irritate the pleura and to cause either a dry pleurisy or a pleurisy with effusion. A superficial cavity may rupture through the visceral pleura (usually during a fit of coughing) and in this way cause a direct tubercular pleurisy which will sooner or later become secondarily infected. A tubercular infection of the pleural membranes may develop during the progress of pulmonary tuberculosis without rupture of a cavity. It is unlikely that such infection is due to a direct spread of organisms, but it is more probable that the bacilli reach the pleura by the blood stream, irritation of the membrane by the neighbouring pulmonary disease being a predisposing factor.

There is no intention in this book of discussing the various hypotheses as to the routes of infection and the relative frequency of them, but it may be noted that the lung is usually infected either by inhalation, by lymphatic infection or by the blood stream. To judge by the behaviour of tuberculosis elsewhere, it is extremely probable that transmission by lymphatic channels is important in conveying the bacillus to the lung. This view is supported by the known frequency (as determined by X-rays) of primary hilum infection. The suggestion of such evidence is that the bacillus, at any rate frequently, enters the mucosa of the upper respiratory tract under the favouring influence

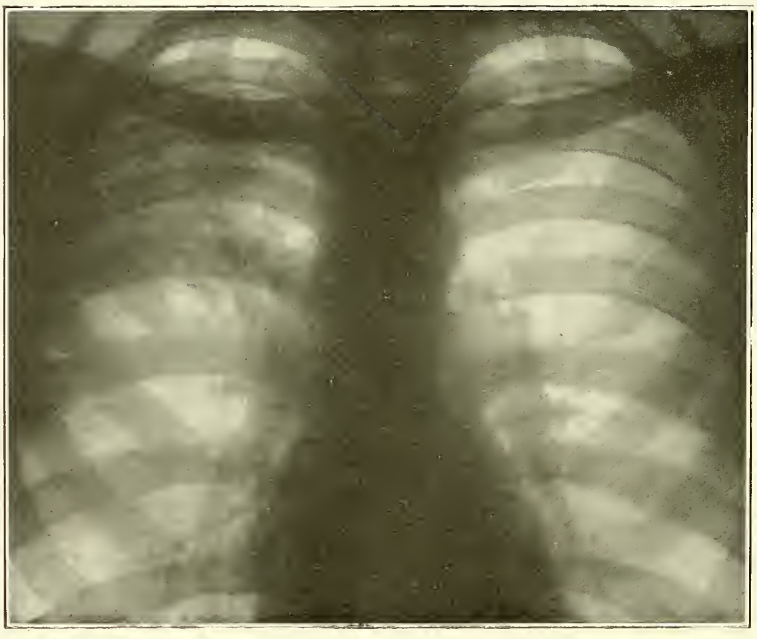


Fig. 43. Tubercular disease spreading from the hilum into the right upper lobe and to a less extent into the middle lobe. Note the complete absence of disease at the apex of the right upper lobe.

of transient lesions there, such as the common cold, and finds its way to the mediastinal lymphatic structures whence it spreads, probably also by lymphatic paths, to the lung. It is to be remembered that while the local inflammatory reaction to the common cold is defensive to the organisms of that disease, it probably lays the affected tissues more than usually open to the attacks of other organisms against which, of course, no specific immunity is being manifested by the local inflammatory reaction. In other words, an inflammation caused by and specifically protective against organism "A" is not only not at all defensive against organism "B" but renders the tissues more open to invasion by it.

For a long period of time it was maintained that the commonest and earliest seat of infection was in the upper lobe, one to two inches below the apex. This view is still held by many, but radiology has shown that it is comparatively rare to find an isolated area of early tuberculosis in this situation, and that the common site of origin is the region of the hilum. The tubercle spreads thence as a peribronchial infiltration extending gradually into the substance of the lung and showing a very strong preference for the upper and middle as opposed to the lower lobe. This striking difference between the clinical and radiological findings is not so incomprehensible as at first sight appears. The peribronchial invasion shows a preference, as has been said, for the bronchi supplying the upper lobe, it produces changes in the walls of those bronchi tending at first to diminution of the lumen and an irritation of the mucous membrane, with a consequent increased activity of the glands. These changes, starting in the deeper parts of the lung, are not directly audible with the stethoscope, but as they necessarily affect the free entry of air and the conduction of sound to the peripheral (and apical) part of the lung, where signs are more easily discovered, it is understandable that a peribronchial lesion may be ascribed clinically to the upper part of the upper lobe.

CLINICAL PHENOMENA.

The clinical phenomena of phthisis are, except in the very acute cases, at first symptomatic only (lassitude, loss of appetite, wasting, slight fever and cough). Very occasionally the first evidence of the disease is hæmoptysis. The later ones depend on the rate of spread of the tubercle, on the fibrosis in the lung and the mechanical changes resulting therefrom ; on the degree of involvement of the various surfaces of the pleura (the formation of adhesions) ; on the necrotic changes in the granulomata (cavity formation) ; on the acuteness of the secondary infection ; and also on the extra-pulmonary complications, such as rupture into the pleural cavity, direct infection of the pleural membranes, involvement of mediastinal and other glands, the spread of tubercle to other parts of the body (laryngitis, peritonitis and meningitis), and toxic myocardial degeneration.

The symptoms and physical signs are dealt with so fully in medical text-books that it is unnecessary here to describe them in detail, but it must be remembered that, just as the pathology varies in different individuals and at different stages of the disease, so do the symptoms and physical signs. There are two points to which special attention may be called : (1) The toxicity of the tubercle bacilli is extraordinarily low, and while it may produce a considerable rise in temperature, the constitutional symptoms which are associated with it are comparatively few. When, therefore, the constitutional disturbance is considerable, it is an indication of the absorption of toxins produced by the secondary infection rather than by the primary one. (2) There is a group of cases in which the symptoms are cough with a certain

amount of expectoration in which no tubercle bacilli can be found, wasting, very considerable lassitude, some pyrexia and symptoms of chronic gastric disturbance. There are signs of slight fibrosis in the lungs. It is extremely hard to determine the diagnosis in such cases. Some of them are undoubtedly tubercular in character; in others, it is probable that the whole of the disturbance has a gastric origin.

There are certain errors in diagnosis which must be referred to. The commonest of these is, undoubtedly, the failure to recognise the disease in the incipient stage, with the result that the patient may lose many months before treatment is begun. Once tubercle is definitely established the tendency is to under-estimate the extent of the disease, particularly when it is peribronchial and deep-seated.

A diagnosis of pulmonary tuberculosis is sometimes made when the lesion is a non-tubercular fibrosis, a bronchiectasis, an infection by a streptothrix or a syphilitic gummatous infiltration.* It is not justifiable to make a definite diagnosis of tuberculosis and to start the patient on a long course of treatment when tubercle bacilli cannot be found in the sputum in repeated examinations, unless the clinical evidence is indubitably corroborated by radiography.

In the earlier stages of the disease, before the granulomata have undergone degenerative changes, it may be extremely difficult or impossible to find the tubercle bacilli in the sputum. With the change in the character of the lesion due to necrosis, communication is established between the bronchi and the interior of the broken down granulomata. Tubercle bacilli, in addition to pyogenic and other organisms, will now, as a rule, be found in abundance. But it must be remembered that in some cases the primary tubercular infection has become arrested and yet the symptoms and signs of cavity formation, of fibrosis, and of toxic absorption persist. This persistence is, of course, due to the continued prevalence of the secondary infections which, having been enabled to invade the lung by the destructive action of the tubercle bacilli, have outlasted their allies and continue to wage the war with unabated vigour.

RADIOLOGY.

Radiology is of extreme importance in the diagnosis of phthisis and is absolutely essential before laying down a rational scheme of treatment in any given case. The importance of the X-ray examination is due to the fact that without it it is impossible to be certain of the extent, and sometimes of the position of the lesion, and even occasionally of the diagnosis.†

* The presence of symptoms out of all proportion to the physical signs, the copiousness of the sputum, the existence of syphilitic lesions in other parts of the body, or the appearance in a radiogram of a single or multiple smooth rounded shadows, or of a hilum and peribronchial fibrosis, when there are no tubercle bacilli in the sputum, are all suggestive of syphilis and indicate the necessity for a Wassermann test.

†Despite the importance of radiology, it is not for one moment suggested that this method is to be used in preference to others, but rather as an essential part of the clinical examination.

The various morbid changes in the lung which have already been described (*i. e.*, granulomata, fibrosis, cavity formation, pneumonic consolidation and bronchial dilatation) are all recognisable by radiography. The granulomata, when discrete, appear as small irregular rounded shadows along the course of the bronchioles. A general fluffiness in the structure of the lung is suggestive that the disease is fairly acute and active, while radiating lines indicate fibrosis and attempts at healing. Very little reliance should be placed, however, on the radiographic appearance in estimating the activity of the disease. The only reliable evidence of this are the physical signs.

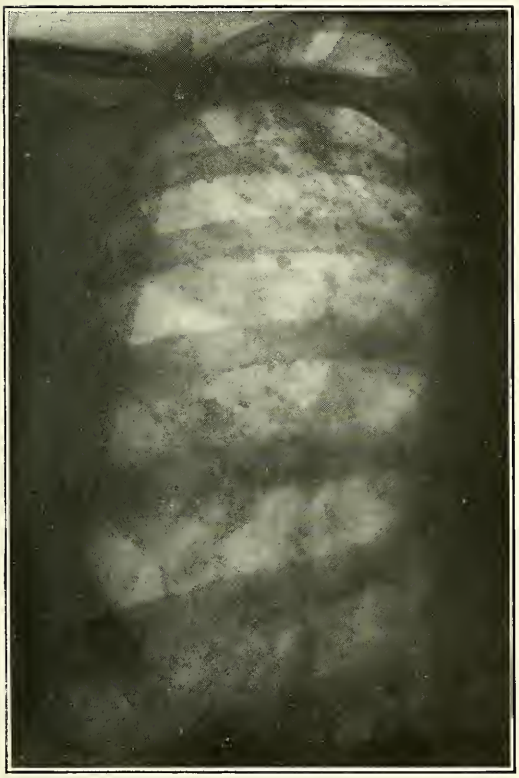


Fig. 44.

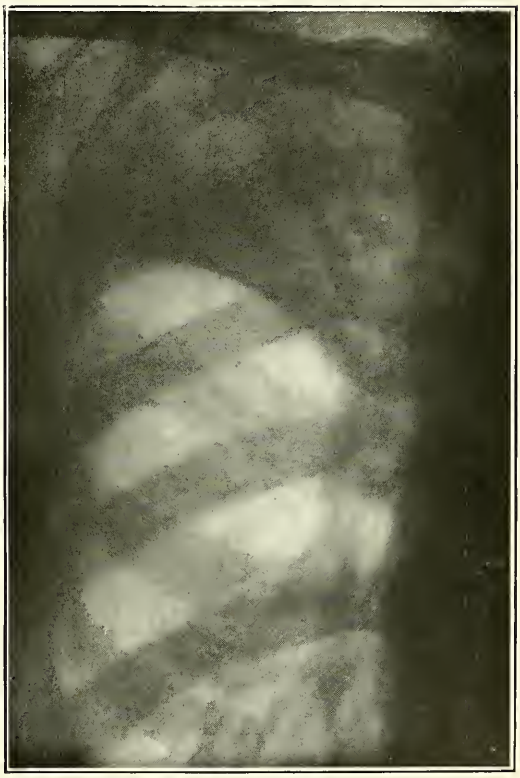


Fig. 45.

Fig. 44. Phthisis of the upper and middle lobes. The supraclavicular portion of the lung is the least affected. The disease is in an active state. Broncholiths are present.

Fig. 45. Tubercular disease of the right upper lobe.

The fibrosis appears in the earlier stages as a fine branching meshwork among and around the tubercular masses, whilst the shadows of the bronchi, due to the peribronchial change, are deeper and broader than normal. When the disease starts as a peribronchial infiltration, the whole hilum appears as a dense mass with branching radiations spreading thence into the various

lobes, those going to the upper being usually the most marked. Enlarged glands, giving opaque rounded shadows, are frequently seen in the region of the root of the lung. In the later stages, when the fibrosis is intense, the whole of one or more lobes may be involved, and all details are obscured by a deep shadow somewhat irregular in density with a very sharp line of demarcation between it and the adjacent part of the lung. Proportionate to the degree of the fibrosis is the displacement of the mediastinum to the affected side, and the increase in obliquity and approximation of the ribs with consequent narrowing of that side of the chest. The appearances due to the fibrosis associated with bronchial dilatation will be dealt with later.

Cavities when large and dry have a well-defined, rounded outline. The interior of the cavity resembles closely a pneumothorax; that is to say,

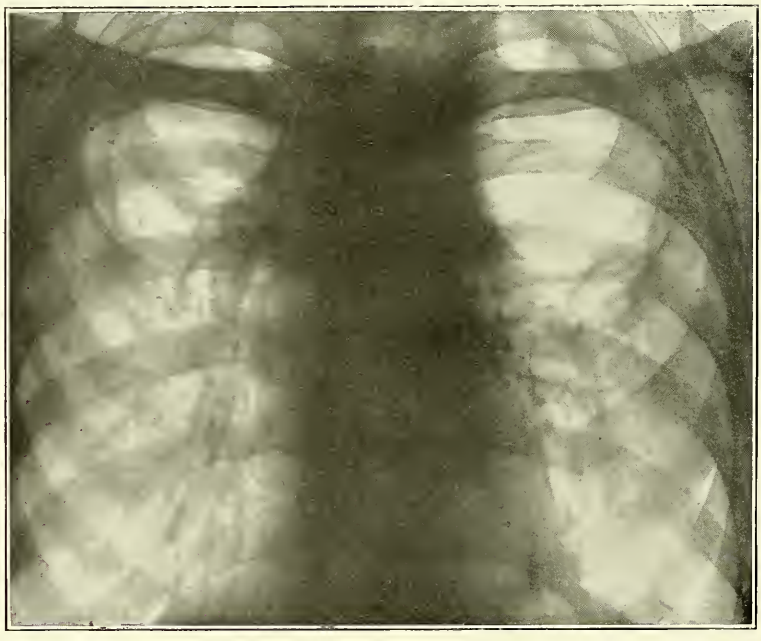


Fig. 46. Chronic tubercular disease of both lungs and mediastinitis. On the left side the upper lobe consists of a single large cavity; there is so little lung tissue left that this area is almost as translucent as a pneumothorax. There is a large cavity in the right upper lobe also, but the antero-posterior diameter of it is considerably less. There is a loculus immediately below the clavicle.

it is abnormally translucent, but shows traces of lung structure unless it extends from one surface of the lung to the other. When recent and acute, the outline is visible, but is less distinct and is irregular. If empty, it is more translucent than the normal lung, but less so than a pneumothorax, and some lung structure is visible. The breadth (antero-posterior) and length and the presence of loculi can be determined by the degree of the translucency.

The appearance around a cavity of a zone of homogeneous opacity with an irregular margin is an indication of surrounding pneumonic consolidation, the result of secondary infection.

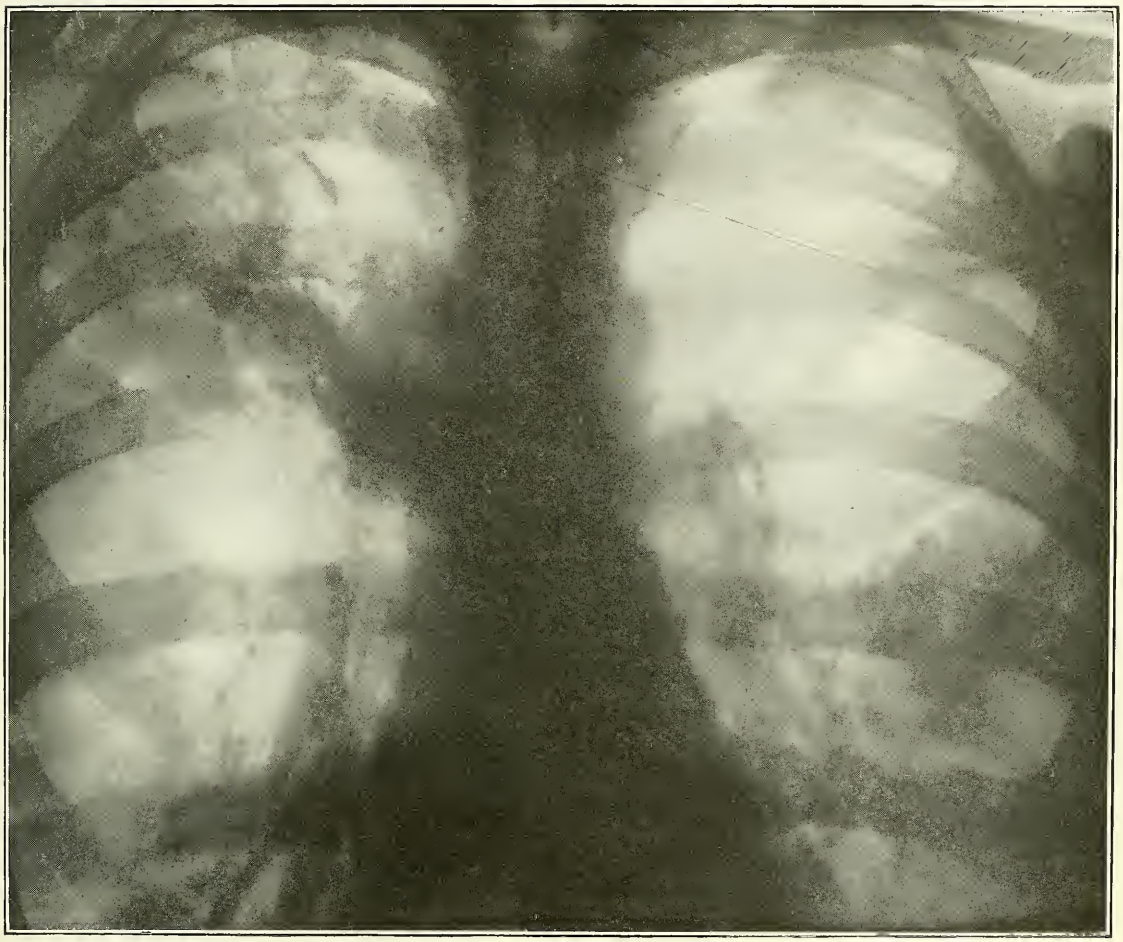


Fig. 47. Tubercle of the right upper and left lower lobes. On the right side there is a cavity surrounded by a very irregular zone of pneumonic consolidation. Enlargement of the glands in the region of the hilum in both sides.

A typical advanced case of phthisis complicated by bronchiectatic dilatation is shown in Figs. 48 and 49. The radiographic appearance of such a case resembles closely that seen in some cases of bronchiectasis (*cf.* Fig. 67). There is the same change in the chest wall, the same extensive displacement of the mediastinum and the same uniform dense shadow involving the lower part of the lung. In the radiogram of bronchiectasis the whole lung is opaque, but the density diminishes gradually from below upwards. There is, however, no other irregularity in the shadow, no lung

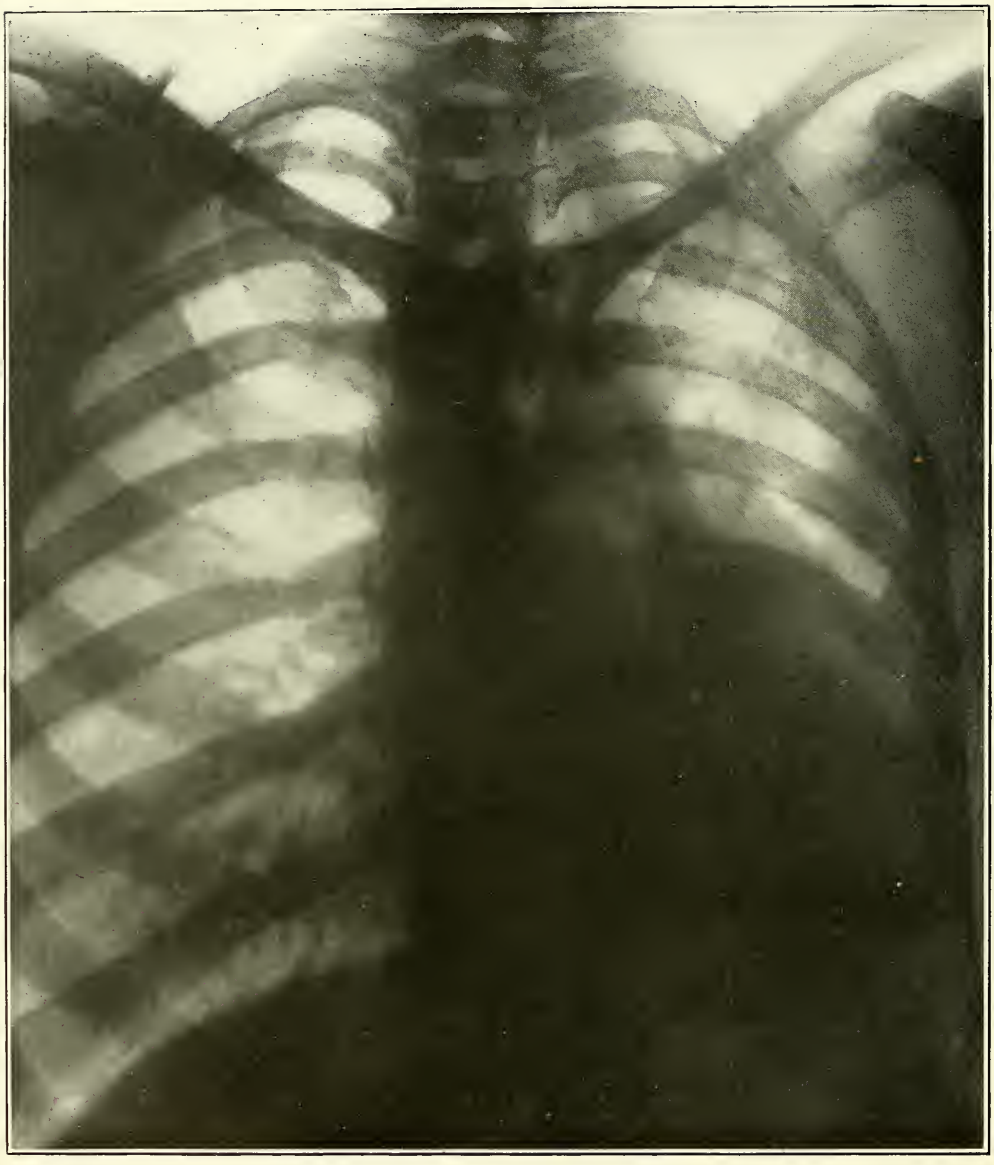


Fig. 48. Tubercular disease of the left lung. Extensive fibrosis and secondary bronchial dilatation. Note the displacement of the heart and of the trachea to the affected side. (*Cf.* Figs. 49 and 67.)

structure is visible, and the whole has a ground-glass appearance. In the radiograms of phthisis, on the other hand, some details of the disease are visible (in the one case the irregular nodular shadows, and in the other a cavity) and the homogeneous ground-glass opacity characteristic of bronchiectasis is absent.

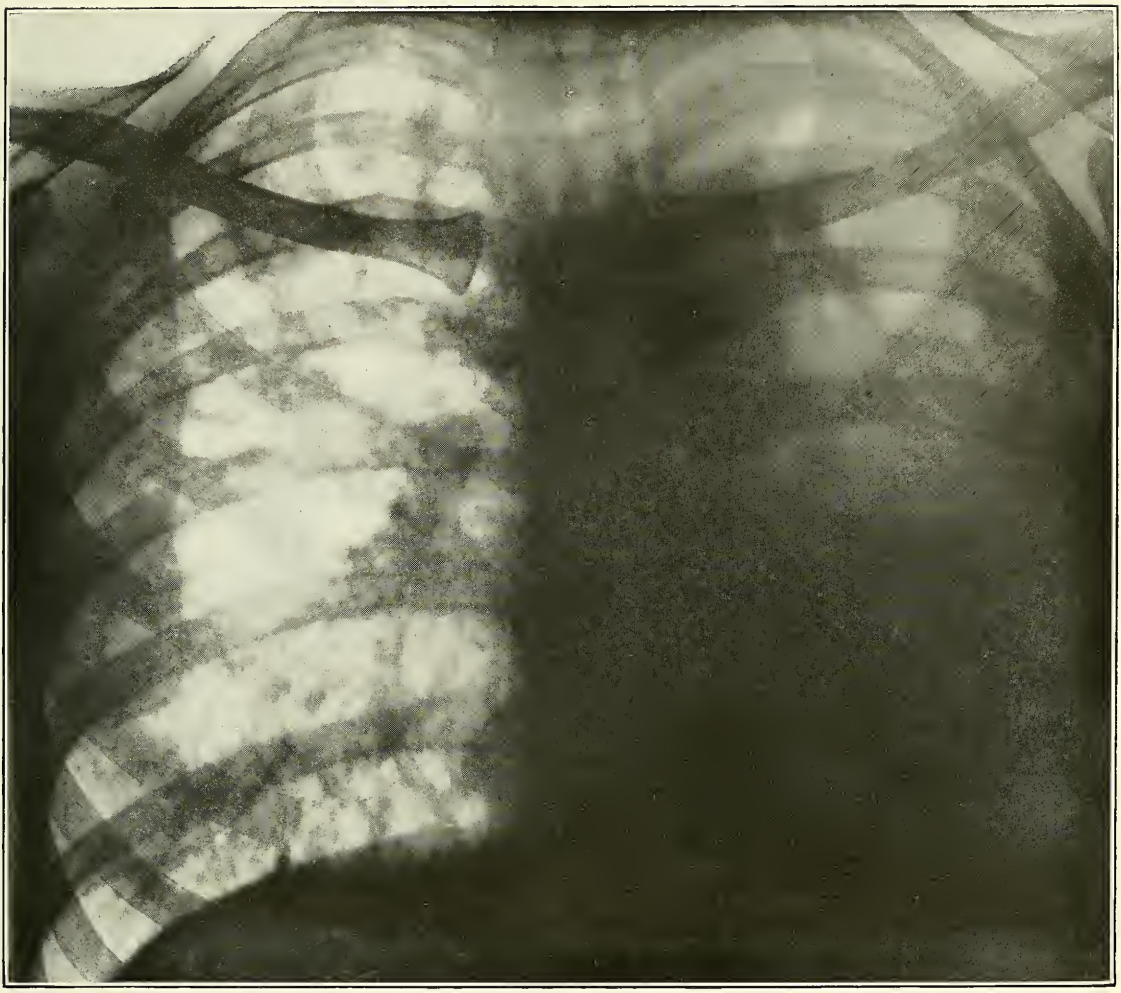


Fig. 49. Bilateral phthisis. On the left side the disease is chronic. There is very considerable fibrosis and secondary bronchial dilatation. There is a cavity in the upper lobe. On the right side the disease is more recent and acute. (*Cf.* Figs. 48 and 67.)

The existence of adhesions between the costal and the visceral pleura cannot be ascertained by radiology, unless there is some gas or fluid in the pleural cavity causing a partial separation of the membranes (Figs. 56, 57 and 58). Adhesions between the lung and diaphragm produce, in the course of time, alterations in the shape of the muscle, which is dragged upwards by the pull of the contracting lung transmitted through the adhesions (Figs. 56 and 67).

A radioscopic as well as a radiographic examination should be made in every case. With the screen it is possible to obtain a general impression

of the lesion, but details are often missing and the examination is limited in time. Certain information is obtainable, however, by this method alone, especially the extent of or absence of movement of the diaphragm, and the degree of "lighting up" of the various parts of the lung during a full inspiration. The plate, if the exposure is correct, and if it is taken with a soft tube, and if there has been no movement of the lung owing to breathing during the exposure, will show not only the gross lesion but the detail also and allows the case to be studied at leisure from its radiological aspect.

Radiology is of great importance in the diagnosis and essential before determining any active line of treatment. As will be seen later, it is invaluable also as a method of control during the treatment, and it is the only means of obtaining accurate knowledge of the conditions and changes in the pleural cavity during nitrogen displacement.

TREATMENT.

Treatment is most effective when based upon full pathological interpretation of the clinical state. The extent to which this familiar principle can be pushed in dealing with pulmonary tuberculosis is perhaps not always taken full advantage of, and there is a considerable tendency to regard the disease as a unit capable of a standardised uniform treatment and to ignore the immense range of variation individual cases present. Routine sanatorium treatment has as its principal object the improvement of the general power of resistance of the body, and tuberculin dispensaries direct their energies to increasing the local resistance of the tissues : both are instances of this selective attitude. When the numerous complications which occur in pulmonary tuberculosis are considered, and the special importance of most of them, it becomes obviously impossible to obtain the maximum beneficial results if all patients are treated in a stereotyped manner.

It may be said that every case of tuberculosis has its own individuality, no two resembling each other in their signs and in their reaction to the disease and response to treatment. This individuality must be studied.

The abnormal features which may call for intervention are numerous. The grouping and the relative importance of them varies with each patient.

Treatment may be required :—

(*a*) To improve the power of resistance to the tubercle bacilli.

(*b*) To place the granuloma under conditions most favourable to its success.

(*c*) To compensate for the mechanical disabilities which have developed as a result of the fibrosis. To prevent the development of bronchial distortion and dilatation, or, if already established, to neutralise its deleterious influences. To obliterate existing cavities.

(*d*) To prevent, if possible, secondary infection, or if present, to reduce absorption of the toxins.

(*e*) To control hæmorrhage and to guard against its recurrence.

(*f*) To abolish or diminish the distress which may be occasioned by certain symptoms.

In addition to these, intervention may be necessary for extra-pulmonary complications, especially (1) of the pleura, (2) of the larynx and pharynx, (3) of the abdomen (including the anal canal), (4) of the heart and kidneys in cases of acute and long continued toxæmia.

The treatment of some of these conditions is entirely medical, while of others it is necessarily surgical. Since indications for both methods of treatment may be present in the same case, it is an unwise and unscientific policy to segregate these defensive measures. The medical and surgical methods of treatment are in no way alternative schemes; they should be worked in collaboration since each mutually assists the other. It is necessary, therefore, to review briefly the medical means of intervention in addition to considering in greater detail the surgical possibilities, so that the value and the limitation of each may be appreciated: so that also it may be understood how surgery attains its greatest use in supporting medical treatment, without replacing it, and in occupying the gaps in the medical lines of defence.

(*a*) *The improvement of the general power of resistance.* This is obtained by hygienic measures, by graduated exercise, by regular hours of rest and by nutritious diet The object is to increase the general tone by a system of training, in principle similar to the training of athletes, but each stage is naturally much more gradual and more cautiously approached. A constant watch is kept on the temperature, as this is the simplest guide to the reaction of the tubercle, a sudden rise indicating that the process of training is too rapid.

The increase of weight obtained by the intensive method of feeding is apt to mislead and is not necessarily beneficial. Adipose tissue does not mean increase of tone but rather the reverse, as fat is likely to be deposited at the same time in other regions of the body, notably the heart.

The effect of gradual training is always beneficial in improving the general health, but such improvement does not necessarily imply that the tubercular disease is in process of being arrested. A patient may acquire and maintain for a time a most excellent physique without any diminution being apparent of the signs of activity in the lungs.

On the other hand, this treatment may be all sufficient, but it is so in the early stage of those cases only in which the natural resistance against the infection is normally high, but has been temporarily reduced by unfavourable conditions of life. When the natural resistance of the patient to the tubercle bacilli is much below that of the average man, improvement of the general tone, while of immense assistance, is inadequate as a method of

cure, as is shown by the frequency with which patients break down after returning to ordinary life.

In no case, however good the natural resistance of the body, can the sanatorium be in itself capable of completely curing the disease, once the mechanical disabilities have increased beyond the power of the individual to compensate for them by natural means—*i.e.*, once the disease has passed what is called the "early stage."

There is one point in connection with this general treatment which is too often overlooked. In a considerable number of people there is a constant absorption of toxins and possibly of micro-organisms from chronic foci of infection in the teeth, in the gums and in the upper respiratory passages. So long as these foci exist, they must necessarily interfere to a greater or lesser extent with the general health of the patient. The eradication of these sources of infection is therefore essential as a preliminary to any other form of treatment.

(*b*) *The need for placing the granuloma under conditions favourable to the successful exercise of its curative function.* It has just been stated that in no case in which the natural resistance is low can the improvement in the general tone do more than produce a temporary improvement. When the patient returns to the previous unsatisfactory environment, the advantages gained by the special course of training will gradually disappear and the disease will resume its interrupted progress. It is evident that some special means of reinforcement is necessary. The granuloma is subject to constant movement during respiration and to spasmodic and violent movement during coughing. These movements are prejudicial to its function. If intrapleural adhesions are absent, it is possible to produce complete collapse and immobilisation of the lung by nitrogen displacement. This treatment is in principle similar to the fixation and immobilisation of a tubercular joint or tendon sheath.

(*c*) *The compensation of the mechanical disabilities which are the result of the fibrosis.* During the early stages of fibrosis, the diminution in volume of the affected part of the lung can be compensated for by the approximation of the surrounding walls and by emphysema of the healthy lung tissue. By the time these changes have reached their limit, the more serious consequences of fibrosis will be developing (bronchial distortion and dilatation and secondary infection).

In a chronic tubercular cavity the surrounding fibrosis and the rigidity of the walls enclosing the lung prevent the obliteration and the healing of the cavity,* The patient is always exposed therefore to the dangers of secondary infection and of hæmorrhage.

* It is true that a cavity which has free drainage may dry up and become symptomless. There is, however, always the risk of re-infection.

These mechanical disabilities are controllable by surgical measures only. It is obvious that if a considerable reduction in the volume of the lung can be brought about, these mechanical adverse factors cease to be of serious importance. The fibrosis no longer exerts its adverse influence on the bronchi and bronchioles; the walls of the dilated bronchioles and of cavities in the parenchyma collapse, retention of secretions is made impossible, the dangers of secondary infection are reduced and those of hæmorrhage become non-existent. The object of surgical intervention is to produce collapse of the lung.

(*d*) and (*e*) *The control of secondary infection and of hæmorrhage.* Cavities in the lung are the most important predisposing cause of these two conditions and the treatment of these cavities as indicated in the preceding paragraph is, therefore, the most efficient means of dealing with the complications.

Either secondary infection or hæmorrhage, however, may become such a predominant feature of the disease that it is necessary to concentrate on the means of controlling it. A secondary infection of both lungs will not respond to surgical measures. A partial control may be obtained by autogenous vaccines. The response to this treatment is variable; in some cases it is extremely gratifying.* When the infection is unilateral and is directly associated with one or more cavities, collapse of the lung will not only control the infection but will prevent its recurrence.

A hæmorrhage will, as a rule, respond to drugs, especially morphia. If the hæmorrhage is persistent, is constantly repeated, or a further loss of blood would endanger the patient's life, the lung should be collapsed by nitrogen (if in cases of bilateral tuberculosis it is known from which lung the hæmorrhage comes). Adhesions may prevent complete collapse, but even a partial displacement is, as a rule, sufficient.

The treatment of symptoms, of the complications involving the larynx and pharynx, the abdomen, the heart and kidneys, do not call for any detailed discussion in this book, but it must always be remembered that such complications must be taken into full consideration before deciding on any line of active treatment. The complications affecting the pleural membranes have been dealt with fully in Chapter IV.

SURGICAL TREATMENT.

No direct attack on the diseased area of the lungs is attempted by the present day methods of surgical treatment. The older forms of treatment—drainage of cavities through the chest wall, etc.—have been proved to be unsatisfactory. The object of the more modern methods of surgical

* It must be emphasised here that no attempt is being made to deal fully with the various alternative methods of medical treatment, viz., in this case, free iodine, inhalation of coal tar products, etc.

intervention is to produce a collapse of the whole or of part of the lung. The results obtained by such treatment are :—

(*a*) The immobilisation of the diseased lung.

(*b*) The abolition of the mechanical disabilities produced by the pull of the contracting fibrous tissue on the mediastinum, on the diaphragm and on the chest wall, and also on the walls of the bronchi.

(*c*) The obliteration of abnormal cavities and of the abnormally dilated existing spaces (bronchi) by the approximation of their walls.

(*d*) The diminution, by the changes in the cavities just referred to, of the secondary infections and the prevention of retention of secretions.

(*e*) The prevention or the arrest of hæmorrhage.

(*f*) As a result of the above changes, the immediate diminution or even the abolition of the main symptoms of the disease. Of these, the diminution in the cough and the pyrexia due to pyogenic infection are the most gratifying, as these symptoms contribute so greatly to the state of ill-health from which some patients suffer.

There are four methods by which collapse of the lung may be obtained.*

(1) Nitrogen displacement (artificial pneumothorax).

(2) Rib mobilisation (Wilms operation, modified).

(3) Local replacement by tumours or foreign bodies.

(4) Partial collapse of the lower lobe by paralysing the diaphragm (section of the phrenic nerve).

Nitrogen Displacement.

Nitrogen displacement is the method of choice in all cases for which collapse of the whole or the greater part of the lung is required, but the success of the treatment is limited by the presence of or by the extent of the intrapleural adhesions. The technique is extremely simple once the details have been mastered ; but it requires considerable experience, since every case varies in the reaction of the disease to the displacement, in the amount of nitrogen that can or should be run in at one time, in the resistance to the displacement caused either by adhesions or by pneumonic or fibrous changes

* No reference is made in the text to the operation for the complete removal of the lung. The cases in which it is possible are so extremely rare and the operation is so dangerous, that it is not to be compared to the more modern methods. Nor is reference made to Freund's operation for division of the first costal cartilage. The original observations which formed the basis of the suggestion of this treatment were insufficient and a fuller investigation of the conditions failed to corroborate them.—"A Consideration of the Influence of the First Costal Cartilage on Apical Tuberculosis." H. Morriston Davies. *British Journal of Surgery*, Vol. 1, No. 1, 1913.

in the lung, also in the mobility of the mediastinum and in the rate of absorption of the gas. Nitrogen displacement is without danger if the ordinary precautions are taken. If sufficient displacement cannot be produced to make it worth while to continue the treatment, no harm has been done. No distress is caused to the patient by this procedure. It is only occasionally that it is necessary to confine the patient to bed during the treatment. The period of convalescence is greatly reduced once the displacement is complete, and the patient can return to his original environment and to his work with much less risk to himself and to the community than if treatment had been confined to improving the general state of health only.

The selection of cases requires considerable care. The indications for the treatment are based on the type of the disease, whilst the contra-indications are determined by the extent of it.

The indications for nitrogen displacement in pulmonary tuberculosis are :—

(1) Extensive fibrosis affecting chiefly one lung.

(2) Secondary bronchiectasis of one lung.

(3) A single cavity, 2 cm. or more in diameter.

(4) Multiple cavitation in one lung.

(5) A single continued, or recurrent hæmoptysis.

(6) Retention of secretions on the one side.

(7) All cases of mainly unilateral disease which progress in spite of medical treatment.

The contra-indications are :—

(1) The absence of sufficient healthy lung tissue on the opposite side to carry on the functions of respiration (*i.e.*, an involvement equivalent to more than one-third).

(2) The presence of cavities or of retention of secretions in the opposite lung.

The lung on the side on which the nitrogen displacement has been done will benefit directly by this treatment. The effect on disease in the opposite lung depends on the extent and the character of the changes. If the lesion is a localised peribronchial one and the mechanical changes are not pronounced it is probably a secondary extension from the side in which the infection originated and will improve *pari passu* with the subsidence of the primary focus of the disease. The treatment of the one lung will exert a less pronounced influence on the disease in the other lung when the mechanical changes on that side also are sufficient to produce symptoms.

Technique of Nitrogen Displacement.

The patient should be under close observation for five to seven days before the first injection is made. The amount of the daily expectoration is recorded and the temperature is taken every four hours during the day.

Nitrogen displacement is without danger and it is painless provided that certain precautions are always taken.* The dangers are syncope from pleural reflex and gas embolism from the introduction of the nitrogen into the veins of the lung. These two conditions have been dealt with at length at the beginning of Chapter V. The syncopal attack, which may last a few seconds only or terminate in the death of the patient, is a reflex which is started by the puncture of the sensitive pleura with the needle. Sharp pain is also produced by the irritation due to the passage of the needle through the parietal membrane. Some patients are much more sensitive than others, and very few can prevent an involuntary movement at the moment when the needle pierces the pleura, and any movement, however slight, is sufficient to interfere at the onset with the injection of gas into the space between the pleural membranes. This pain and the pleural reflex can be entirely abolished by anæsthetising the track of the needle. The method by which this is done is described on page 46, and the various details of the technique should be fully understood.†

Certain further precautions should also be taken on the first, and possibly the second, occasion of injecting gas into a patient. These are the administration of a third of a grain of morphia‡ and the introduction of at least 50 c.c. of oxygen before any nitrogen is run in.

When the point of the needle is in the potential space between the two pleural membranes, a negative pressure is registered on the manometer, but the respiratory variations are so slight that it is at times an open question whether the opening in the needle is in the pleural cavity or in the lung. As soon as the pleural membranes are separated by a layer of gas, the difference between the expiratory and inspiratory pressures becomes quite clear. If the point of the needle is in the lung and gas is run in, there is always the danger that some of it may be forced into the pulmonary veins and be carried into the terminal arteries of the brain (gas embolism). The occurrence of a cerebral lesion has never been recorded when oxygen is used as the avidity of the tissues for the gas is such that it is probably all absorbed before it reaches the brain.

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* Even without these precautions, the danger is slight, but the risk is absolutely unjustifiable, and the pain at each puncture may jeopardise the success of the treatment.

† The technique is extremely simple, but it is seldom that sufficient trouble is given to the various details, and in consequence the patient frequently suffers pain and runs the risk of a reflex syncopal attack.

‡ The morphia lessens the apprehension of the patient, is in itself of value in reducing the risk of pleural reflex, and ensures a greater degree of immobility during the delicate manipulations necessary for the introduction of the first 100 c.c. of gas.

There are very many different forms of apparatus in use for introducing nitrogen.* In some, the gas is generated as required, whilst in others, there are glass containers which are filled from cylinders of compressed nitrogen. The essential features of all should be a water manometer, a filter and some means of gently displacing the nitrogen from the containers into the pleural cavity. The manometer should be so connected with the rest of the apparatus that readings can be taken throughout the operation.

The author's apparatus is shown in Fig. 50. There is a large graduated container for the nitrogen and a smaller one alongside for the oxygen. At the beginning, the air in these two is driven out by the fluid from the other

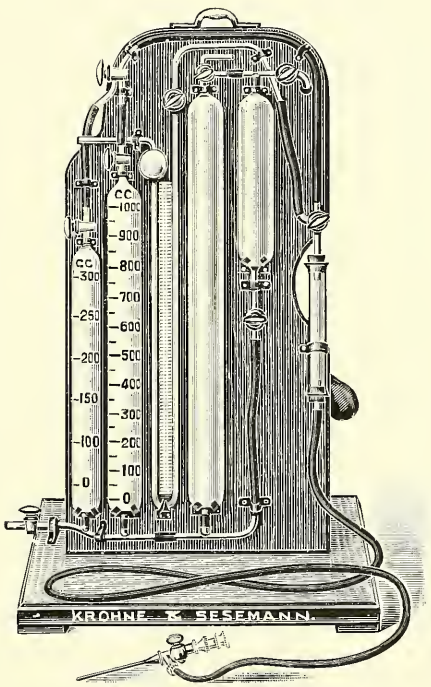

Fig. 50.

two containers. They are then connected with the compressed gas cylinders, and the fluid in the one is replaced by nitrogen, and in the other by oxygen. Between the needle and the gas there is a sterilised filter and a three-way tap. Before the needle is inserted into the chest wall, this tap is turned so that the manometer only is in connection with the needle by way of the filter; but before the gas is run in, the tap is turned for a quarter of a circle which brings not only the manometer but the gas also into connection with the

* Nitrogen is used as the rate of absorption of it is extremely slow as compared to oxygen. Further, nitrogen is the gas which normally replaces oxygen in the pleural cavity. (See footnote, p. 54.)

needle, and so with the pleural cavity. When air is allowed to enter through the tap at the top of the water container, the water, seeking to find its own level, flows into the graduated container, displacing the gas into the pleural cavity. The rate and the amount of the flow is controlled entirely by this tap.

The shaft of the needle is made on the principle of a Potain's needle, but the needle itself and the stilette resemble those used for spinal puncture. The bore is 1 mm. in diameter, is open at the extreme end and there is a second aperture at the side. The shaft is graduated in centimetres. The needle, the tube leading to the filter, and the filter itself are all sterilised before use.

The most convenient spot for puncturing the chest wall is between the anterior and posterior axillary folds in the 5th, 6th, 7th or 8th spaces. In this region the bony framework is not covered by continuous muscles and the spaces therefore are easily distinguished from the ribs (see Fig. 4). The patient should be in a semi-recumbent posture, well propped up by pillows, with the arm supported across the chest or above the head.

When there are no adhesions between the pleural membranes, the amount of gas run in on the several occasions in order to collapse the lung is determined by the reaction of the patient to the last injection, as measured by the rise of temperature ; but even when there is no reaction, the amount should never be more than 200 c.c. at the first, 350 c.c. at the second, 500 c.c. at the third and 750 c.c. at the subsequent injections until the collapse is complete. When adhesions are present, the amount of gas injected is determined entirely by the pressures.* It is quite impossible to decide as to whether the lung is completely collapsed or not except by radiography. The patient is not necessarily confined to bed during the treatment. If prior to the injections the health of the patient has been sufficiently good to allow him to be up, he should not be kept in bed except for about twenty-four hours after each of the first three injections, and for a few hours after the subsequent ones, provided there is no reaction. Should there be a reaction, the patient must remain in bed until twenty-four hours after the temperature has subsided, and for a somewhat longer period than that advocated above after the subsequent injections.

Nitrogen Displacement in the Absence of Adhesions.

The following is a detailed description of the technique of nitrogen displacement in a patient whose pleural cavity is free from all adhesions. Apparatus required : A Record syringe, a long hypodermic needle and 2 per cent. novocaine, a double-edged tenotome, the nitrogen apparatus including the needle, nitrogen and oxygen gas.

* The question of intrapleural pressures has been discussed in some detail in Chapter II.

1st injection. A third of a grain of morphia is given twenty minutes beforehand. The large and small graduated containers are filled with nitrogen and oxygen respectively, the sterilised needle and filter are attached, and the apparatus is tested to ensure that it is working efficiently. The patient's skin in the axillary region is painted with iodine and an area of skin and of the deeper tissues down to and including the pleura in the 6th or 7th intercostal space is anæsthetised by infiltration with novocaine (see p. 46). The skin in the centre of this area is punctured with a double-edged tenotome* and the needle is driven slowly and carefully through the intercostal space, the stilette is withdrawn and the tap in the handle of the needle closed. The depth of the parietal pleura is usually about 2·5 cm.; it is sometimes less, but, if the patient has much subcutaneous fat, is always more. The moment the opening of the needle reaches the pleural cavity, a negative pressure equal to from 4 to 7 mm. of mercury will be recorded on the manometer. Slight variations corresponding with the respirations should always be observed. Deep inspiration will produce a temporary increase in the negative pressure, and a cough a sudden slight decrease.

At this stage the slightest movement of the patient or of the operator will displace the point of the needle. If a negative pressure is registered on the manometer but no variations are seen, it will mean that the needle has passed through into the lung, or having entered the pleural cavity has slipped again outside the parietal membrane. In the former case, a deep inspiration may produce a further increase in the negative pressure, which will not, however, return to its former level. The needle should be withdrawn and the stilette passed through it, as the opening is frequently blocked when it has entered the lung. A further attempt must then be made to get and keep the point of the needle between the membranes. As soon as this is accomplished, oxygen is allowed very slowly to enter the pleural cavity, until about 50 c.c. have been introduced. The fluid in the manometer should now show very distinct variations in level corresponding with expiration and inspiration. If these are present, there can be no doubt that the point of the needle is between the parietal and visceral membranes, and there can be no longer any danger of a gas embolism. Nitrogen is therefore run in until the pleural cavity contains 200 c.c. of gas (oxygen 50 c.c., nitrogen 150 c.c.). The intrapleural pressure variations are noted, the needle is withdrawn and the small wound covered with a piece of zinc oxide plaster about 2 cm. square. This plaster, which is the only dressing required, must be removed on the ensuing day, otherwise the retained moisture in the wound will favour infection from the skin.

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* The skin offers great resistance to the needle of the nitrogen apparatus. The wound made by the tenotome allows the needle to be driven steadily through the anæsthetised tissues and prevents the sudden rush on to a rib or into the lung, which is so often seen when the needle is forced through the skin. It obviates also the risk of the conveyance into the deeper tissues of organisms from the skin.

2nd injection. (*a*) *When there has been no reaction as the result of the first injection* (*i.e.*, when the temperature has not risen higher than on the preceding days), the second injection is given after forty-eight hours. 350 c.c. of nitrogen are run in. There is no need of morphia or of oxygen, but the track of the needle must be anæsthetised with novocaine. The needle is passed through the same interspace close to the previous puncture.

(*b*) *When there has been a reaction* (*i.e.*, when the temperature has risen higher than on previous occasions), the second injection must not be made until the day after the temperature has returned to or below its previous level. Not more than 250 c.c. of gas must be run in ; less if the reaction has been a big one. If there has been a delay of five or more days, the operator will experience much the same difficulties as at the first injection. Morphia may therefore be given if thought advisable, and oxygen must certainly be injected before the nitrogen—that is to say, the procedure adopted on the first occasion is repeated in its entirety.

3rd and subsequent injections. (*a*) *When there has been no reaction.* An interval of two days must be allowed to elapse between each injection and the track of the needle must always be anæsthetised with the greatest care. 350 c.c. of nitrogen are introduced on the third occasion, 500 c.c. on the fourth, and 750 c.c. on the fifth and subsequent occasions until the lung is completely collapsed.*

The total amount of nitrogen required to produce complete collapse of the lung in an adult varies from 3,500 to 8,000 c.c. This immense variation depends not so much on the variations in the size of the pleural cavity, as on the activity of the patient. If during the treatment the patient is kept confined to bed the lower figure will be approximately correct. When, however, the patient leads a fairly active existence, the rate of absorption is much more rapid, and therefore a much larger amount of gas will be required to obtain the complete collapse.

(*b*) *When the injection causes a reaction.* Whenever there is a rise of temperature as a result of an injection, no more gas must be run in until the reaction has completely subsided. The increase of the amount of nitrogen introduced on each subsequent occasion must be determined by the extent of the reaction. If there has been a rise of temperature of over 2 degrees, the amount of nitrogen at the next injection should be the same as on the previous one ; but if of about 1 degree, an additional 100 c.c. of nitrogen may be run in. No special precautions other than those mentioned in the paragraph above are required.

The puncture for each of these injections should be through the same intercostal space or the next one to it. If on any occasion there is difficulty in finding the interpleural interval, the attempt must

* It has already been stated that total collapse of the lung can be ascertained with certainty only by the X-rays.

be postponed and the patient X-rayed. The difficulty may be due to adhesions, to the re-expansion of the lung, to the presence of an effusion, or to faulty technique.

Fig. 51 is a characteristic chart of the variations of intrapleural pressures which occur during the collapse of the lung by nitrogen displacement. With increase of the amount of gas, there is a gradual widening of the difference

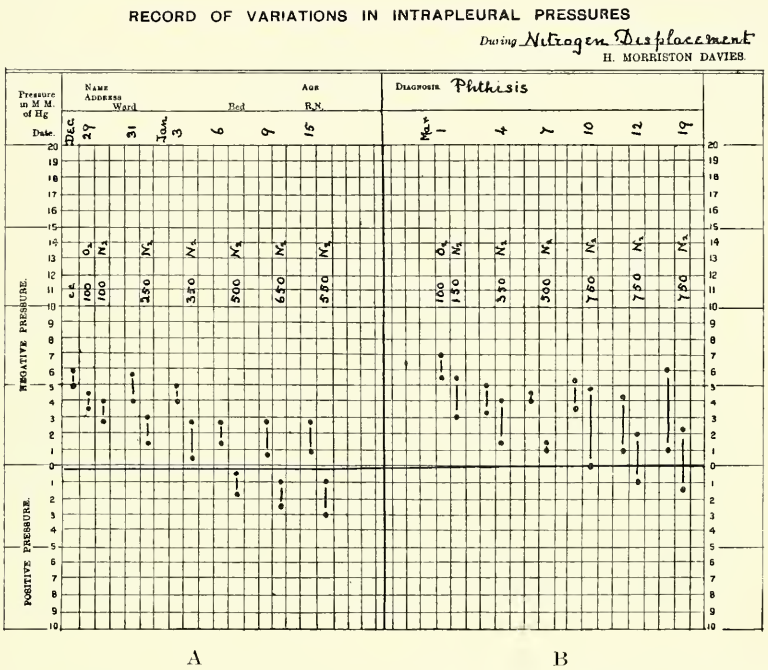

Fig. 51. The records of the variations of the intrapleural pressures during the displacement of the lung by nitrogen in the absence of pleural adhesions. The inspiratory and expiratory pressure before and after each injection of gas is shown.

between the inspiratory and expiratory pressure, but this is present so long only as there is a free pleural cavity. The pressure at the end of each injection is less negative than at the beginning, and this decrease in the negative pressure becomes more marked as the collapse of the lung nears completion. At the beginning of an injection, the pressure is usually more negative than it was at the end of the previous one, but less so than at the beginning of it, unless a pleural effusion has developed in the meantime.

When a diseased lung is displaced, whether by nitrogen or by a spontaneous pneumothorax, the lung is collapsed as a flattened band along the length of the mediastinum (see Fig. 25). A healthy lung collapses, however, on to the hilum and resembles a pedunculated rounded tumour. The reason for the difference in the two cases is, probably, that in the latter

the elasticity of the organ draws the various parts of the lung down to the root, whilst in the former case the rigidity of the bronchi and the fibrosis interfere with the free contraction of the elastic tissue.

The Immediate Results.

The sputum, and to some extent the cough, are at first increased as a result of the injections, but after some 2,000 c.c. of nitrogen have been let in, there is a rapid decrease of these symptoms, and, when the lung is collapsed and the disease is unilateral, they disappear entirely. In such cases also there is a corresponding change in the temperature ; there may at first be an

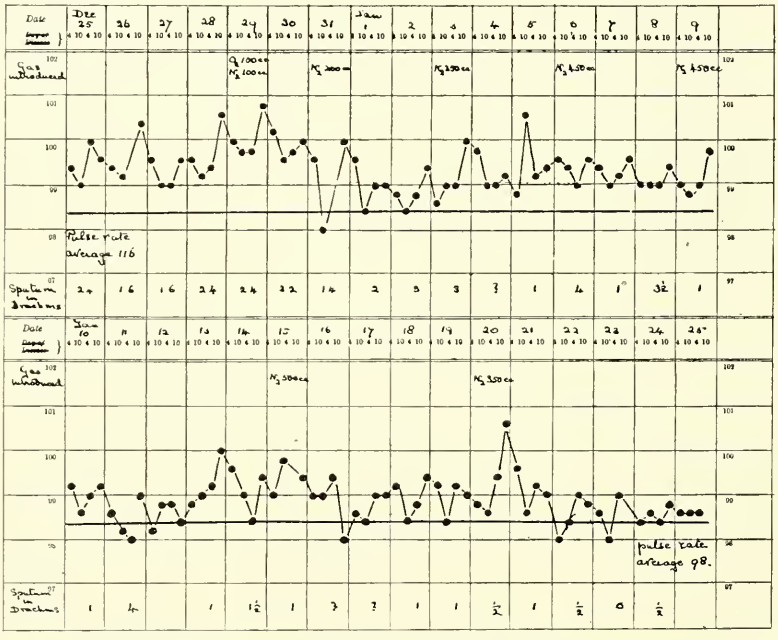

Fig. 52. Temperature chart of a patient before, during and after the displacement of the lung by nitrogen. The effect of this treatment on the sputum and on the pulse rate is also shown.

elevation as a result of reaction, but at the end of the immediate treatment the temperature becomes normal. Since there is no longer retention of secretions the absorption of toxins is reduced to a minimum, and since also the patient is no longer distressed by his cough, and possibly by want of sleep, the general health begins to undergo rapid improvement.

There is, during the period of the production of the collapse, some loss of weight, but this is soon regained and during the subsequent months there is generally a progressive increase. If previously there was hæmoptysis, it will cease.

These changes occur also, but to a less striking extent, when there is disease also on the opposite side.

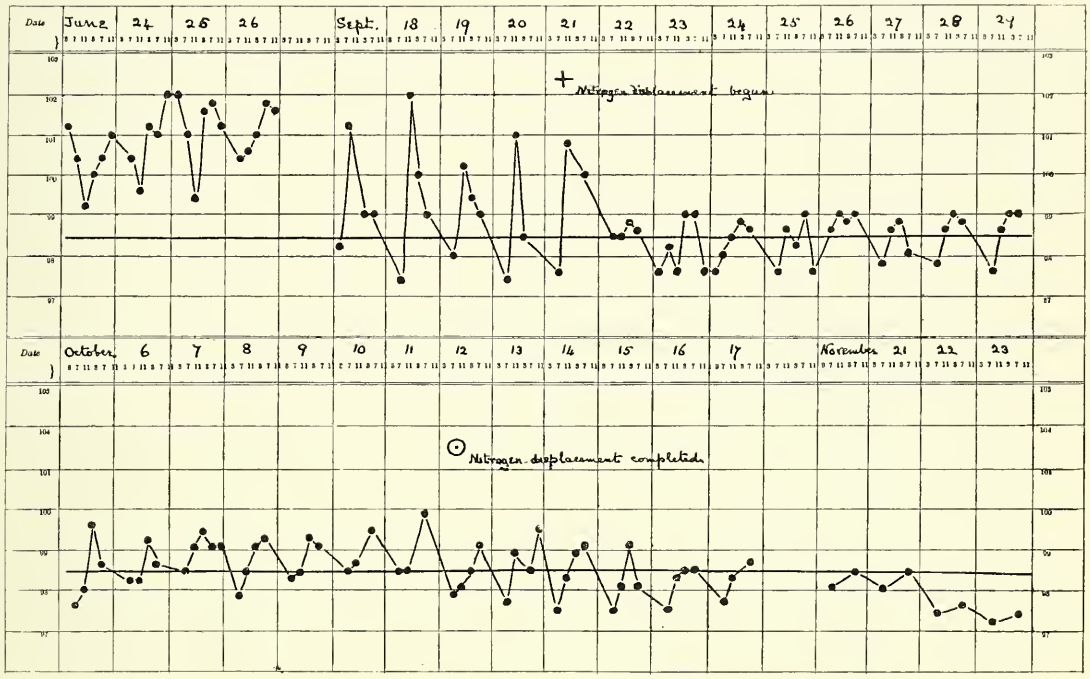

Fig. 53. Temperature chart of a patient before, during and after the displacement of the lung by nitrogen.

After Treatment.

From the moment that the collapse of the lung has been completed by the final injection of nitrogen, there is an extremely gradual re-expansion of the organ as the result of slow but steady absorption of the gas. This has to be checked by replacing the absorbed gas periodically by nitrogen. As the object is to keep the lung in a state of complete collapse, it is obvious that the more frequently the pleural cavity is refilled with nitrogen, the more nearly is the object attained. Experience has shown that during the first four months at any rate these refills should be made, if practicable, every two or three weeks.

The rate of diminution in the volume of gas in the pleural cavity is dependent in part on the ability of the lung to re-expand. When a diseased lung has been collapsed for a considerable period of time, the fibrotic changes which have taken place limit greatly its expansile capacity, and a comparatively very much higher negative pressure is required to draw it out than is necessary for a normal lung, or even for a diseased one recently collapsed. It is for this reason that after the first few months the rate of

absorption of the nitrogen appears to be less rapid than in the earlier period,* and the refills need be made less frequently. The period of time which should elapse between fresh injections can be gauged most accurately by noting, by means of the X-rays, the change in the position of the mediastinum and lung during a given period of time. As a general rule, it may be stated that the refills should be made every three to four weeks during the second period of four months, every five to six weeks during the third period of four months, and so on until such time as it is thought safe to allow fuller activity to the lung.

It is not possible to determine beforehand for what length of time it will be necessary to maintain the lung in a state of complete collapse. This depends in part upon the type and extent in the disease of the collapsed lung; in part upon the efficiency with which the collateral treatment is carried out; in part on the capacity of reaction to the disease by the patient; in part also upon the improvement or progress of the disease (if any) in the opposite lung.

When there is no disease in the opposite side, and the lesion in the lung undergoing direct treatment has been fairly extensive, the collapse should be maintained for a period of two years. After the lapse of that time, no further refills are made, and during the ensuing three to five months the lung will very slowly re-expand.

A return of any symptoms necessitates immediate careful examination. In certain groups of cases in which there has been a secondary bronchial dilatation, a persistence of some symptoms is bound to take place. These are due directly to the changes in the bronchi, and consist of cough and a mucoid expectoration. They are likely, moreover, to become much exaggerated if the patient has a cold, and to persist for some time. In such a case the sputum is definitely muco-purulent. These manifestations, particularly if the previous condition of the lung is known, must not be regarded as a recurrence of the tuberculosis. Until the lung is completely expanded and the chest can be examined with a stethoscope, a diagnosis of the recrudescence of the primary disease can be made on the positive findings of the tubercle bacilli in the sputum or the recurrence of the original symptoms with toxæmia, and on shadows in the radiogram suggestive of active tubercular foci. At the first indication of the recurrence of definite tubercular symptoms (or signs), the lung must be collapsed once more and maintained in that condition for another year at least.

The absence of any indication of active tubercular disease in the lung, when the organ has been allowed again to expand, does not necessarily mean that the patient is cured. The tubercle bacilli may have been eradicated,

* It is improbable that there is any actual variation of the rate of absorption of gas in any individual person. There is, however, considerable evidence that there is a constant interchange of nitrogen and CO_2. If samples are taken at intervals, slight variations in the proportions of these two gases will be noted.

and the patient may be fit to resume a normal life, but there is always a probability that the disease, though dormant, is still present, and that the organisms have become attenuated and enclosed by fibrous tissue. In such cases, if during the ensuing months the patient's health declines as a result of overwork, constant fatigue, excessive indulgence, or some illness like influenza, during which proper care is not taken, the organisms may be able to re-establish their ascendency, to break through the scar tissue about them and start again on their course of destruction. It is, therefore, essential that the patient be warned of this possibility; he should be examined once every three months for at least a year after the cessation of active treatment.

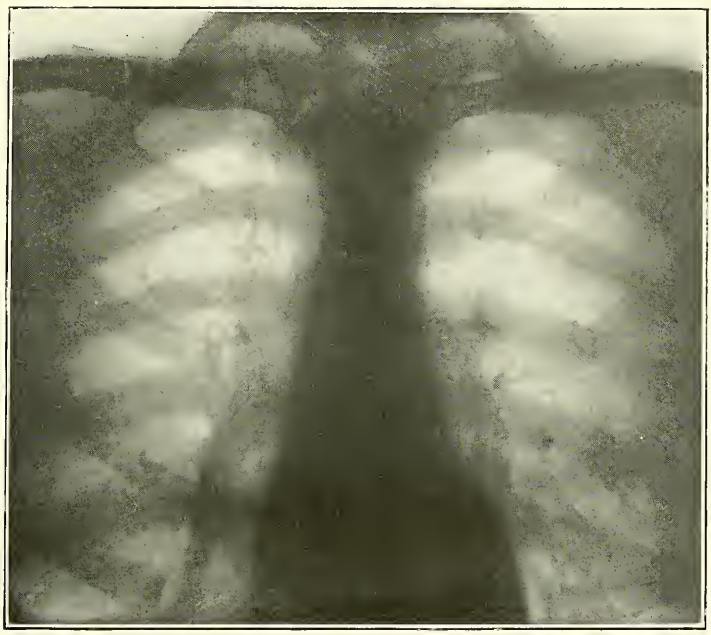

Fig. 54. Healed pulmonary tuberculosis.

The after treatment of those cases in which there is disease in the second lung also, which shows definite evidence of continuous improvement during the period of collapse of the primarily infected lung, is much the same as that described in the preceding paragraphs. The collapse should, however, be maintained for a somewhat longer period of time.

In certain cases, and especially in those in which both lungs have been infected at much the same time, the disease in the lung not under direct treatment may progress during the period of collapse of the opposite lung. This must be regarded as an indication that the treatment adopted is not likely to be beneficial and should, therefore, be abandoned.

Complications of Nitrogen Displacement in the Absence of Adhesions.

A pleural effusion develops in a certain number of cases.* The effusion may appear during the production of the collapse or at any time during the maintenance of it. It should always be suspected if a less negative (or more positive) intrapleural pressure is found when beginning a fresh injection than was known to be present at the end of the previous one. The amount of fluid is rarely large (300 to 1,000 c.c.), and seldom produces symptoms other than a feeling of weight or splashing. If, however, the fluid accumulates rapidly just after the lung has been completely collapsed and there is already a positive pressure in the pleural cavity, dyspnœa and a feeling of great oppression in the chest, or even of suffocation, will develop. As a general rule it is in these cases only that the removal of the liquid is indicated. The method adopted is the same as that described under oxygen replacement (pp. 46 *et seq.*), but nitrogen is used in place of oxygen. The pressure left in the pleural cavity is slightly less than the patient can tolerate with comfort, so as to allow for some re-accumulation of the effusion. A return of fluid sufficient in amount to necessitate intervention a second time is extremely rare.

The reason for the development of an effusion is commonly thought to be irritation of the pleural membranes : in support of this is the fact that it occurs somewhat more frequently when adhesions are present. It is probable, however, that sepsis may be a contributing factor ; the infection, conveyed by the needle or gas, being too slight to produce an extensive leucocytosis and the formation of pus. Very rarely, nitrogen displacement is complicated by an empyema ; such a disaster is due to some fault in the technique.

One of the most remarkable facts in nitrogen displacement is the almost entire absence of dyspnœa when one lung is completely collapsed. The appearance of dyspnœa in patients undergoing this form of treatment is usually an indication of a sudden increase in the intrapleural pressure due to the development of an effusion, or to the absorption of the gas in the pleural cavity and a partial re-expansion of the lung. This last condition has already been referred to in Chapter III on p. 36, where the suggestion is made that the symptom is the result of a disproportion in the capacity for expansion of the lung and of the chest wall. Some shortness of breath persists during the whole period of re-expansion and continues in a lessening degree for some long time after the visceral and parietal pleura have come in contact. It can be completely abolished by again increasing the intrapleural volume of gas and so collapsing the lung. The shortness of breath, except when the result of a sudden increase in intrapleural pressure, is never sufficient to interfere seriously with the patient's mode of living.

* The percentage of all cases is given usually as 33. This figure is considerably higher than is shown by my records.

Dyspnœa is very occasionally the result of some acute change (*e.g.*, pneumonia) in the opposite lung, or of cardiac or renal failure.

The reactions which may be caused during the collapse of the lung have already been referred to. Miliary tuberculosis may supervene if the collapse is brought about in too rapid a manner, either by the injection of too much nitrogen at any one time, or by giving a further injection before the reaction caused by the previous one has had time to subside.

Nitrogen Displacement as a Means of Checking Hæmoptysis.

This method of controlling hæmorrhage is most effectual. Intrapleural adhesions may, however, interfere with the success of it. The rate of the displacement of the lung by nitrogen depends on the urgency of the symptom.

(*a*) If this treatment is a prophylactic measure or is carried out after the bleeding has stopped in order to prevent recurrence, the injection of gas is done in the manner detailed in the preceding pages.

(*b*) When the hæmorrhage is continuous and is endangering the patient's life, the cessation of the bleeding is of primary importance and the possibility of a reaction should not be taken into account. At the first injection, therefore, 500 c.c. of nitrogen are run in, but if this does not suffice to check the bleeding, the injection of gas must be continued until the hæmoptysis has ceased. The urgency of the case does not, however, permit of the omission of any of the details of the procedure, which is described fully on pp. 166 *et seq.*

The subsequent injections and the amount of gas run in are dependent, in the first place, on whether there is recurrence of the bleeding, and in the second place, on the presence or absence of reaction.* If the hæmorrhage recurs, the injection may be repeated the next day, and on the following day also if necessary. When the hæmorrhage has been checked, the further collapse of the lung is carried out on the same principles as already detailed on pp. 168 *et seq.*, and the after treatment is in no way different to that described on pp. 171 *et seq.*

Nitrogen Displacement when Adhesions are Present.

Until an attempt has been made to introduce the needle into the pleural cavity, it is impossible to tell whether the membranes are completely free, whether they are united by isolated fine or dense adhesions, or whether they are fixed throughout a part or the whole of the pleural cavity. Even the free

* The loss of blood appears to have some considerable influence in preventing the development of a reaction.

entry of gas and a persistence of a negative intrapleural pressure during the first two or three injections does not denote the absence of adhesions ; but an alteration of pressure from negative to positive when the quantity of gas is considerably less than should under normal conditions be easily accommodated, is a probable indication that the two membranes are not completely unattached. In these circumstances, it is always unwise, and even dangerous, to continue the injections unless the intrapleural conditions can be ascertained by the X-rays.

For the sake of clearness of description, the technique of nitrogen displacement and the difficulties likely to be met with will be discussed in connection with the following varieties of pleural adhesions :—

(1) When the pleural membranes are universally adherent.

(2) When the intrapleural space is occupied by a thin layer of recent lymph.

(3) When the adhesions involve a considerable area of the surface of the lung.

(4) When a small area only of the visceral pleura is fixed to the parietal.

(5) When there are a few thin scattered adhesions which are capable of yielding to moderate degrees of pressure.

(6) When a single tough adhesion unites a localised area of the visceral to the parietal membrane.

(1) *When the pleural membranes are universally adherent.*

It is impossible in these cases to get the point of the needle into a space between the two membranes. The only variation in the manometer is when the needle enters the lung ; a negative pressure will then be recorded which increases with every inspiration and shows no fall with expiration.

Attempts may be made high up and low down in the axillary line, below the angle of the scapula or on the front of the chest. A sufficient collapse of the lung to be of any value is, however, seldom obtained when the membranes are adherent in the axillary region.

(2) *When the intrapleural space is occupied by recent lymph.*

There is a type of case in which an unusual group of phenomena of infrequent occurrence is met with. The pleural cavity is found only after great difficulty, and the respiratory variations are extremely small. When

oxygen is run in, the manometer shows a rapid change of pressure from negative to positive until it is equal to about 4, 6 or 8 mm. of mercury. The pressure then ceases to rise while the gas continues to enter quite readily. It will be found further that during the subsequent injections, the gas cannot enter except at a considerable pressure, but once this pressure is reached there is no further difficulty.

What the pathological condition of the pleural membranes is in these cases is not known for certain, but the obstruction to free air entry is not due to fibrous adhesions. I would suggest that the membranes are held together by a layer of solid lymph exudate which may be in an early stage of organisation.

(3) *When the adhesions involve a considerable area of the surface of the lung.*

The needle may enter the pleural cavity at once, or only after repeated efforts in different spaces. As the gas enters, the pressure rises rapidly, and when from 50 c.c. to 100 c.c. have run in, the pull on the adhesions causes pain. An attempt is made in another region, and a second space between the pleural membranes is possibly found, but the same limitation is experienced. If the maximum amount of gas which can be run in at any one spot before pain is produced is 200 c.c., it is not worth while to continue this line of treatment. If, however, some 400 or 500 c.c. of nitrogen can be tolerated in two or three different areas, and an X-ray shows that the adhesions are thin and capable of stretching, it is justifiable and advisable to continue the attempt to displace the lung.

The method adopted in such cases differs somewhat from that already described. The rate of flow of nitrogen should be extremely slow, and the pressure must not be raised higher than equal to +12 mm. of mercury unless a deliberate attempt is being made to rupture some of the weaker adhesions.* This attempt to rupture adhesions is permissible only when the gas containing spaces are seen by the X-rays to be shut off from each other and from the rest of the pleural cavity by fine strands of adhesions (Fig. 56), and when, further, it is clear that at no part is the surface of the lung yielding to the strain.

Experience shows that a partial collapse of the lung (if the organ is reduced to at least half its volume), while not so efficient as total collapse, may be of considerable value in a patient who, up to that time, has resisted all forms of treatment. The refills must be made every week for the first two months, and if the highest pressure which the patient can tolerate is maintained, a further yielding of the adhesions is produced with corresponding greater benefit to the patient.

* The various methods of dividing adhesions, and the precautions which must be taken, are described later.

It is most important that the chest be examined with the X-rays and the patient be under close observation, as the irritation of the pleura and the likelihood of an effusion is greater than under normal circumstances.

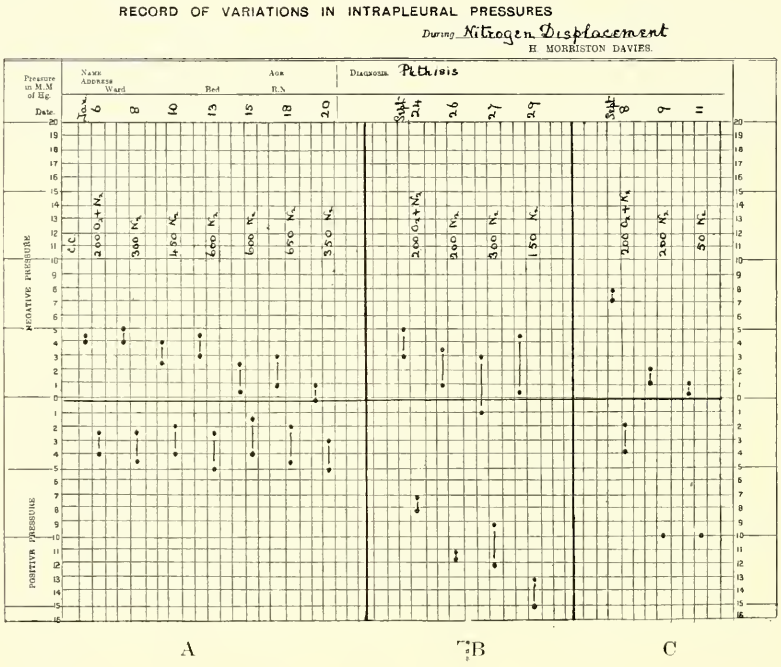

Fig. 55. Charts showing the variations in intrapleural pressure during the displacement of the lung by nitrogen when adhesions were present. (*Cf.* Fig. 51.) In A the pleural membranes were united by recent lymph. In B and C the adhesions were so extensive that treatment was discontinued. The inspiratory and expiratory pressure before and after each injection of gas is shown.

(4) *When a small part only of the visceral pleura is fixed to the parietal.*

The existence of firm adhesions round the apex of the lung whilst the rest of the pleural cavity is free is a fairly frequent occurrence. The lung, instead of being flattened against the mediastinum, is displaced upwards and inwards. The condition should always be suspected when a positive pressure reading is obtained before 3,000 c.c. of gas have been introduced into an adult. It can be confirmed sometimes by the persistence of physical signs in the upper part of the chest, and always by the X-rays. There is no reason for abandoning the treatment because of this localised limitation. Unless there is a cavity in the extreme apex, the results obtained will be very nearly as satisfactory as in a case where there are no adhesions, but the treatment should be continued for an additional six months.

(5) *When there are a few scattered thin adhesions.*

With care and steady maintenance of moderately high pressure (equal to 10, 12 or even 15 mm. of mercury, according to the tolerance of the patient), it may be possible to obtain complete collapse of the lung by stretching or even rupturing these adhesions. The presence of such adhesions is therefore no contra-indication to the continuance of the treatment, but it must be rigidly controlled by repeated examination with the X-rays.

The pleural cavity in these cases is found without difficulty and there is little resistance met with during the injection of the first few hundred cubic centimetres of nitrogen. After this the pressure will rise rapidly, and the subsequent displacement will have to be conducted slowly and with all the precautions mentioned above (p. 177).

(6) *When a single tough adhesion unites a localised area of the visceral to the parietal pleura.*

The resistance to the entry of the gas and the character of the variations in the pressures are almost precisely similar in cases in which there are numerous fine adhesions and in those in which there is a single stout one. The differentiation can be made by the X-rays only, and it is of the utmost importance that this should be done. If the gas is introduced at a moderately high pressure in a case in which the adhesions are slender, they will stretch or rupture before the lung ; but if the adhesion is thick, its stretching power is limited while its breaking resistance is enormous. The first effect of the high pressure will be exerted directly on the lung, the surface of which will be torn close to the attachment of the adhesion. This will result in the production of an acute pneumothorax, hydro- or pyo-pneumothorax. The opening will, moreover, almost certainly remain patent owing to the pull of the adhesion on one margin of it, while the other margin is displaced in the opposite direction by the pressure of the gas or liquid and the tendency of the lung to collapse. Such a condition is illustrated in Fig. 27. The opening in this case had persisted for four months, but became obliterated directly the adhesion had been divided and the lung enabled to collapse.

When, therefore, a single tough adhesion is present, it is never justifiable to raise the intrapleural pressure higher than equal to +5 mm. of mercury.

In the case shown in Fig. 57, a rapid rise in pressure after a partial pneumothorax had been induced indicated that the lung was not collapsing properly, and the X-ray was taken in time to show that a further increase in pressure would, in all probability, rupture the cavity which can be seen drawn out by the attachment of the adhesion to its surface. The only efficient treatment in such cases is the division of the adhesion.

Fig. 56.

Fig. 57.

Fig. 56. Partial nitrogen displacement of the lung. Note the long adhesions stretching from the lung to the diaphragm.

Fig. 57. Partial nitrogen displacement of the lung. There is a strong adhesion passing from the chest wall to the lung immediately over a cavity. For the sake of clearness, the outlines of the adhesion have been emphasised. *Radiographed by Dr. Kincaid.*

The *after treatment* when adhesions are present is carried out on the same principles as have already been laid down for cases in which the pleural membranes are free.

The *complications* also are the same as those mentioned on p. 174, but as has been stated, a pleural effusion is of somewhat more frequent occurrence and may necessitate more urgent measures for the relief of the pressure symptoms. The treatment of pneumo- or pyo-pneumothorax due to rupture of the lung is described on pp. 83 and 67.

There are two other complications which occur only when there are adhesions. They are spontaneous pain and a chronic dry cough due to the irritation of the diaphragm. Pain may develop suddenly during the first

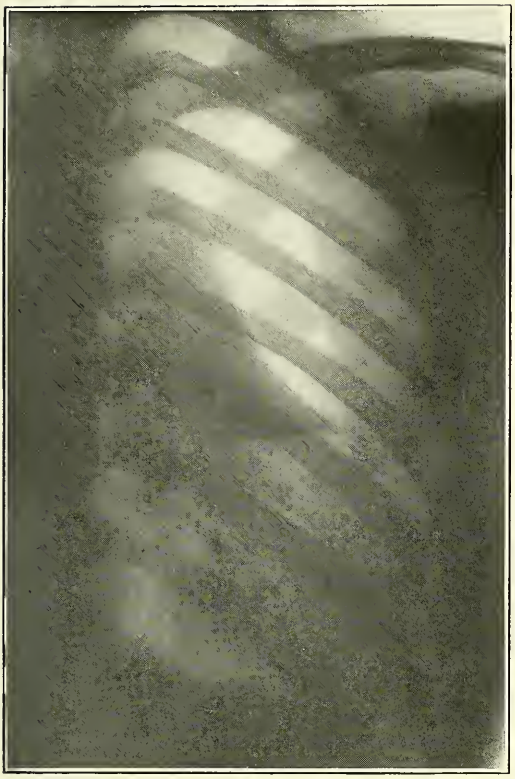

Fig. 58.

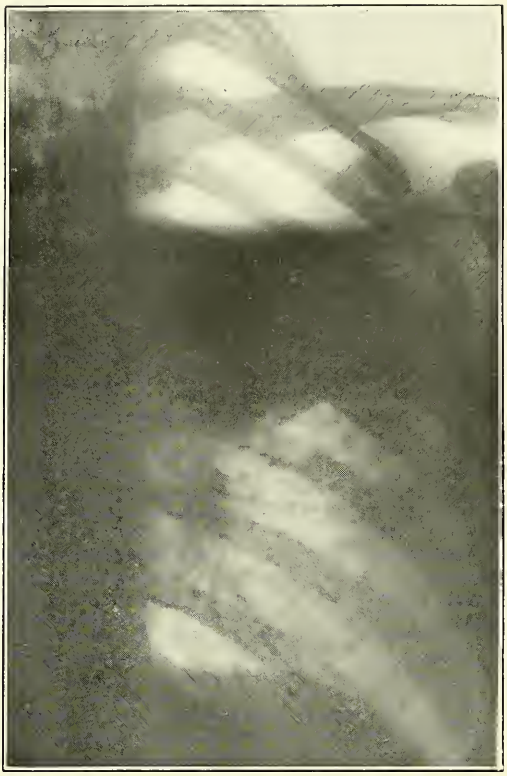

Fig. 59.

Fig. 58. Nitrogen displacement of the upper lobe. The middle and lower lobes are compressed by the pressure.

Fig. 59. Same case six weeks later. The pneumothorax is complicated by an effusion, but owing to the absorption of gas, the intrapleural pressure has diminished.

six to twelve hours after an injection. It occurred in three of my patients but on one occasion only in each case. All three were women and all three had adhesions, and in each case they had been left with a positive pressure. The pain appeared suddenly, the breathing was very rapid, the patient was in every case excited and anxious. The pulse was slightly increased but there was no cyanosis. In none of the cases was there any evidence of the occurrence of a spontaneous pneumothorax or of an effusion, but it was quite obvious that the rapid breathing was aggravating the condition and was to a great extent due to fright. On being reassured that there was no danger, and induced to breath more quietly, the symptoms diminished considerably, and after an injection of morphia the patients went to sleep and woke up free from all discomfort.

A very distressing cough may be caused by the drag on the diaphragm of a collapsed lung, the base of which is fixed to this muscle by adhesions. So long as the patient is absolutely quiet in bed, the symptom is more or less in abeyance, but any movement or exertion is sufficient to produce a dry hacking cough. This cough is so trying that the patient often prefers to stay in bed, though otherwise quite fit to be up and about. If the symptom persists as long as the collapse of the lung is maintained, it is necessary either to divide the phrenic nerve in the neck or to abandon treatment by displacement. The phrenic nerve contains motor and sensory fibres, and the division of it causes paralysis of the diaphragm on the same side, and abolishes also the sensory part of the reflex. The operation is described on p. 192.

Treatment of Adhesions.

Adhesions which interfere with the displacement of the lung by nitrogen, but which allow of sufficient separation by gas of the two pleural membranes so that the character and the extent of the intervening strands can be recognised by radiography, may be dealt with in one of three ways :

1. By rupture of fine strands.
2. By stretching of medium-sized bands.
3. By division of bands which are too stout to yield to a stretching force.

1. *The rupture of fine strands.*—This is quite a justifiable procedure when the bands of fibrous tissue passing from one pleural membrane to the other are so thin that it is certain that they will tear much more readily than the visceral pleura. This point can be ascertained with the X-rays. If with the increasing intrapleural pressure produced by the gas the surface of the lung at the point of attachment of the adhesion shows irregularity in its outline due to the drag of the adhesion, it is dangerous to continue the attempt.

When intentionally* rupturing an adhesion or a series of adhesions, the increase of pressure necessary may cause pain and at the moment that the adhesion breaks the patient feels " as if something had given way "; there is also a very slight risk of pleural reflex. To obviate the pain and the danger of the reflex, morphia should always be given before the attempt is made.

It is rarely wise to raise the intrapleural pressure higher than one equal to 20 mm. of mercury, though I have in one case, in which extreme measures were quite justified, raised the pressure up to 32 mm. of mercury. As the

*Extremely fine strands are occasionally ruptured during the course of a nitrogen displacement without any special increase to the positive pressure.

positive pressure increases, the variations during inspiration and expiration decrease until they may even disappear. At the moment of rupture the pressure drops suddenly and the respiratory undulations of the manometer reappear.

2. *The stretching of medium-sized bands.*—Adhesions which are too tough to rupture may yield to a steady positive pressure equal to 8 or 10 mm. of mercury maintained for a considerable period of time (two or three months). As it is important to keep the pressure constantly high, fresh injections of gas should be given every eight to ten days. The patient should be examined from time to time with the X-rays, so as to make certain that it is the adhesion which is yielding to the stretching force and not the surface of the lung. The case described on p. 179 and illustrated in Fig. 57 is an example of the extreme importance of this precaution.

Adhesions may be made to stretch to such an extent that in certain cases the collapse of the lung which can be obtained is doubled. Unless the peripheral end of the adhesion is attached to the diaphragm, the treatment is usually symptomless, except perhaps for a feeling of tightness within the chest. The pull on the diaphragm may, however, cause pain and an irritating cough. These symptoms will subside with the decline in the pressure.

3. *The division of adhesions.*—A single adhesion, or even two or three which are so stout that they resist an adequate degree of stretching, can be treated by division, but in order to cut the band it is necessary to obtain either a direct or an indirect view of it.

The commonest situation for a single adhesion is between the lower border of the upper lobe and the parietal pleura opposite the 2nd rib either anteriorly, laterally or posteriorly. It is most necessary to obtain a clear knowledge of the situation and of the direction of the band, and for this it must have been possible to separate the two pleural membranes by at least an inch.

Division of a band or bands should not be attempted unless they are seriously interfering with efficient collapse of the lung. The band may be short and broad, or long, narrow and cylindrical. The latter only are accessible for section by indirect vision.

(*a*) *Section of a band by direct vision* is done through an opening in the chest wall close to the adhesion. The band is divided by a knife and the incision through the wall of the thorax is closed. The details of the technique have already been described on p. 59. The bleeding from the band is never serious enough to cause anxiety.

(*b*) *Section of a band by indirect vision.*—A view of the band is obtained by X-ray illumination. The technique of the operation is as follows: "The day or two before, an injection of gas should be given so as to tighten the band by displacing the lung as far as it will go. Two hypodermic injections, each of a quarter of a grain of morphia, are given, the one half-an-hour and the other immediately before the operation. The patient lies on a couch with the X-ray tube beneath, while the assistant holds the screen. The tube should be fitted with a diaphragm so as to limit the rays to the field of operation. It is advisable for the surgeon to wear smoked glasses, which are removed only when the X-rays are turned on. When the peripheral attachment of the adhesion is to the front of the chest, the tenotome is passed into the pleural cavity through an intercostal space in the mid-axillary line, but if the adhesion is fixed to the lateral or posterior aspect, the tenotome is passed through the front of the chest.

The tenotome which I had made for this purpose has the blade 2" cm. long by 0·6 cm. broad, sharpened at both edges, and gradually increasing in thickness to the shank. This part of the instrument is 12·5 cm. long, and has the same breadth and thickness as the base of the knife, but the edges are rounded. It in turn is attached to a short handle. The shank passes readily through the incision in the chest wall made by the blade of the tenotome, but fits so closely into it that there is no escape of gas. The wound through the pleura closes on withdrawing the knife, and unless there is an immediate fit of coughing, there is no subsequent escape of gas into the cellular tissues. The track through which the tenotome passes is previously anæsthetised with novocaine.

As soon as the tenotome is in the pleural cavity, the room is darkened and the X-rays are turned on. Both the adhesion and the tenotome are now visible and the latter is directed towards the former till the edge of the knife rests on the band. The resistance which this offers to the knife can be felt and the movement of the band and of the lung produced by the pressure of the knife can be seen. The adhesion is cut through by sawing movements.

A little blood may escape from the cut edges if the adhesion is a broad one, but the bleeding soon stops, and is of no material consequence. The patient is kept quite quiet for the next twenty-four hours, but beyond this no special after treatment is necessary."*

RIB MOBILISATION.

(*Collapse of the lung together with the chest wall.*)

This operation is reserved for those cases of phthisis in which extensive collapse of the lung is necessary, but in which no, or insufficient, separation of the pleural membranes by nitrogen displacement is possible.

* "The Importance of the Mechanical Factor in the Treatment of Pulmonary Tuberculosis." H. Morriston Davies. *Quarterly Journal of Medicine*, Vol. 11, No. 43, April, 1918.

Much greater care, however, is required in the selection of the cases, as it is impossible, once the operation is done, to obtain re-expansion should there be a spread of the disease in the other lung. Rib mobilisation must not be attempted if there is any active disease in the opposite lung, or if the fibrotic changes in that organ are at all extensive. One of the chief reasons for this contra-indication is that, when one lung is almost entirely out of action and the other is much fibrosed, the work thrown on the heart, weakened already by toxic absorption, is more than it can adequately deal with, and it will sooner or later show signs of failure to continue to overcome the strain. Under the most favourable circumstances, this organ will have extra work to do, and it is necessary, therefore, to take into consideration the state of the cardio-vascular as well as of the pulmonary system when deciding on the advisability of rib mobilisation.

The general condition of the patient is comparatively of little importance ; wasting, sleeplessness, vomiting, while making the operation more hazardous, must not be regarded as contra-indications. Cachexia or evidence of considerable impairment of the heart, or of arterial degeneration, preclude all possibility of success. The risks are not great either at the time of the operation, if done in two stages and if anoci-association is used ; or during the after treatment if certain precautions to be mentioned are taken. Success depends on the correct selection of the case and on the shortness of the interval between the two stages of the operation. The length of this interval is determined by the extent of the reaction of the patient to the first stage.

The object of the operation is to secure a general collapse of the lung by mobilising the whole of one side of the thorax so that the lung and chest wall collapse together. The chest wall is rendered mobile by removing at the first stage a short length from the posterior part of each of the first ten ribs, and at the second stage the costal cartilages of the first and following ribs down to and including a portion of the costal margin.

The *technique* of the operation and the effect of the rib mobilisation on the lung and on the chest wall are given in detail in the next chapter (pp. 212 *et seq.*) ; the procedure is the same whether it is undertaken for bronchiectasis or for phthisis. Certain points in the preliminary preparation and in the after treatment which are of special importance in phthisis are : the radiographic examination of the chest before undertaking the operation ; the delay of five to seven days if the patient has been moved to a nursing home, in order to note the variations in temperature and to allow for the subsidence of any reaction which may have been caused by the journey ; the feeding of the patient with plenty of sugar and the ensurance of sleep at night by morphia, if necessary. After both operations (first and second stage), the bandage should be so adjusted as to give firm support to the chest and to prevent the dressings from slipping, but it must not be so tight that it causes discomfort and necessitates readjustment ; neither bandages nor

dressing should be changed until the stitches are taken out on the tenth day, as the alteration of the support to and the compression of the ribs may cause a fresh reaction.

The second stage must be done as soon after the first as possible. The interval is determined to a great extent by the reaction. It is advisable

Fig. 60. Chronic pulmonary tuberculosis before treatment by rib mobilisation.

to postpone the second operation until the temperature has returned to or below its former level ; but if the rise of temperature persists for more than four weeks, it will be impossible to delay any longer without jeopardising the success of the whole procedure, owing to the refixation by fibrous tissue and callus of the divided posterior ends of the ribs.

The results obtained by rib mobilisation and by complete nitrogen displacement are very similar, since the immediate object of both operations is an extensive collapse of the whole lung. A noticeable improvement in the patient's condition may be observed after the first stage of rib mobilisation, but the main effect will not be obtained until a few weeks after the second

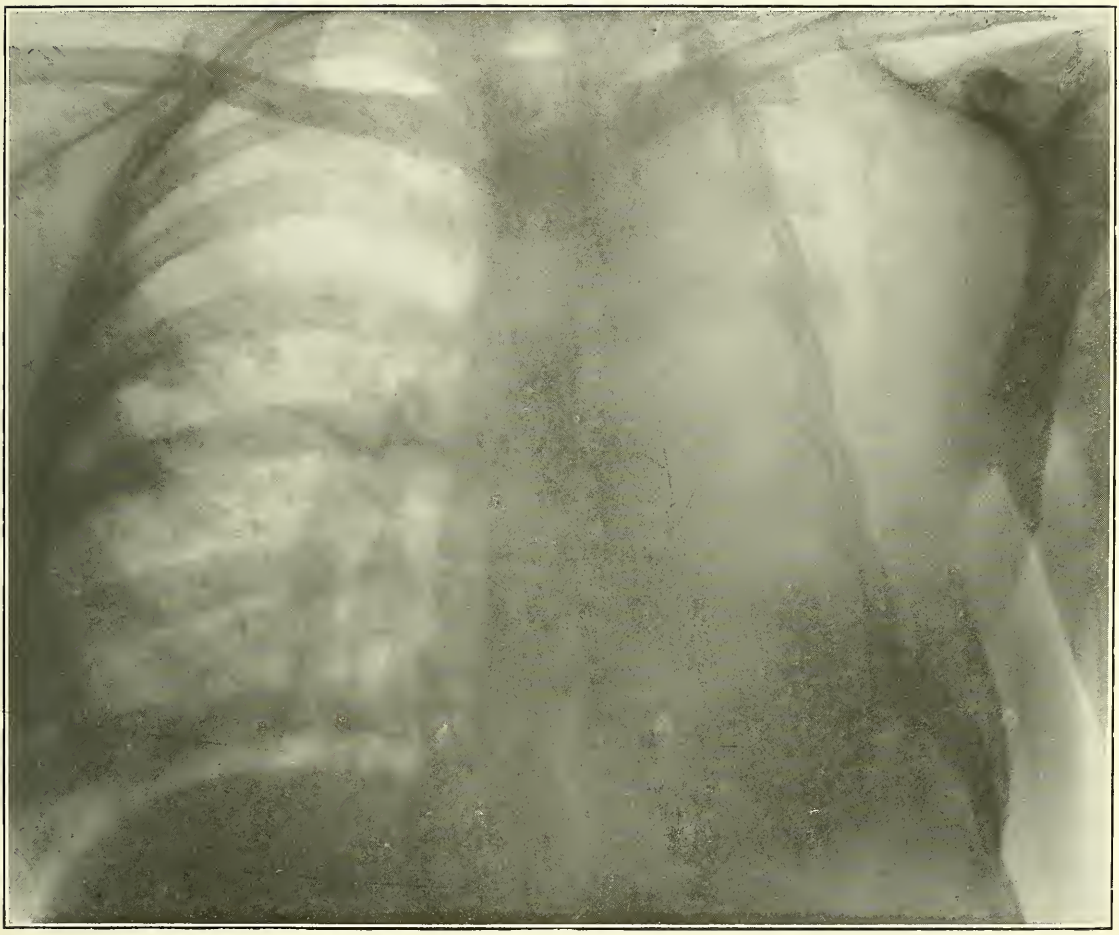

Fig. 61. Same case after rib mobilisation. *Radiograms by Dr. Ironside Bruce.*

stage, by which time the patient will have recovered from the shock and the debilitating effects of what must be regarded as two major operations.

Nitrogen displacement and rib mobilisation should not be regarded as alternative measures ; the latter is justifiable only in a few of those cases in which displacement has been attempted and has failed. Rib mobilisation is a much more drastic form of treatment, and the collapse of the lung and

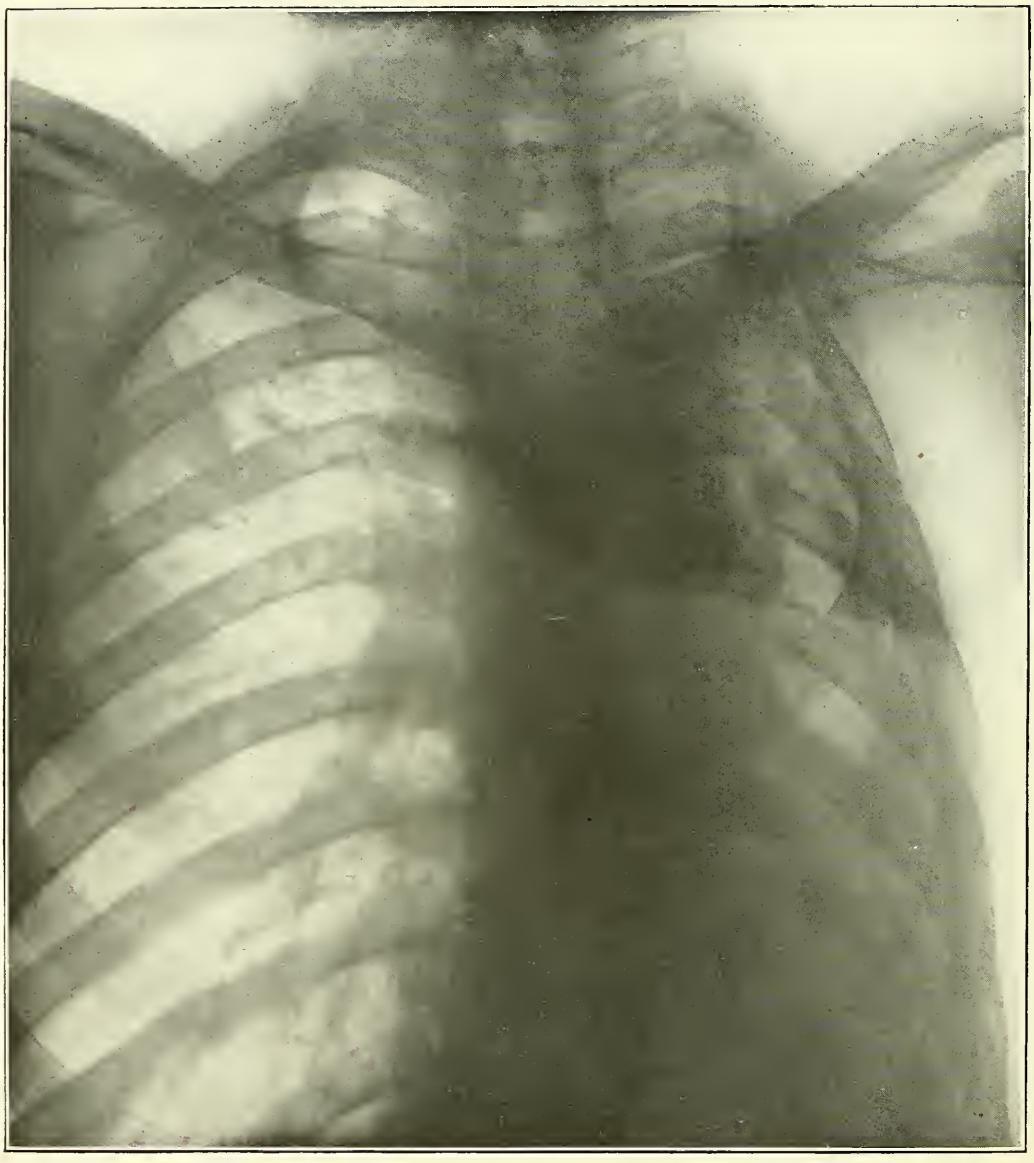

Fig. 62. Pulmonary tuberculosis treated by rib mobilisation.

chest wall are permanent and, should the disease spread into the opposite lung, re-expansion is impossible. It has, however, two advantages—(1) the collapse of the lung, though not quite so complete, is permanent, and there are no variations in the size of the organ as is the case in nitrogen displacement. The granulomata, therefore, are kept even more completely

at rest than by the method of collapse by gas ; (2) when the operation is completed there is no need for further surgical intervention during the ensuing two or three years, as is the case with nitrogen displacement. This does not mean that the treatment of the patient's general health can be neglected. It must be remembered that though the symptoms have greatly subsided, though the granulomata have been placed under conditions most favourable to their success and though the mechanical disabilities have been compensated and the danger of secondary infection abolished, the disease is still present. For the ensuing year, therefore, it will be necessary for the patient to maintain his general state of health at as high a level as possible so as to give the granulomata every possibility of carrying on and successfully completing their normal function.

When the primary disease has been complicated by a considerable dilatation of the larger as well as of the smaller bronchial tubes, there is usually some persistence of symptoms (morning cough and mucoid expectoration), due, not to the action of the tubercle bacilli, but to a chronic bronchitic condition in the main uncollapsed bronchi. This chronic irritation renders these patients more liable to attacks of bronchitis, especially as a sequela to infections such as the common cold or influenza.

Local Displacement by Foreign Bodies.

There is a type of case very occasionally met with in which the disease is limited to the upper lobe only of one side, and in which the mechanical changes (especially cavity formation with retention of secretions) are so advanced as to necessitate operative intervention before there can be any hope of improvement. The displacement by nitrogen may be impossible or inadequate owing to the presence of adhesions, whilst collapse by rib mobilisation would deprive the patient of a considerable area of healthy lung tissue. It is in these comparatively rare cases that the subcostal extra-pleural displacement of the lung by a foreign body may be done with advantage.

The parietal pleura is readily stripped from the under surface of the ribs and can be displaced inwards together with the lung. The space thus caused must be filled by some substance in order to maintain the immobility and the collapse of the upper lobe. Nitrogen is useless, as there is no containing membrane on the outer wall of the artificial cavity. Two types of foreign substances have been used : (1) Paraffin or pastes which have paraffin as a basis ; (2) transplanted living tissues, *e.g.*, lipomata, fibromata or a mass of fatty omentum. Paraffin is not satisfactory, as it may produce irritation and the weight of it is uncomfortable. Tuffier, who is a strong advocate of this operation, recommends the use of living tissues.

In all cases in which active treatment is directed against the upper lobe only and the disease is limited to this part of the lung, it is advisable

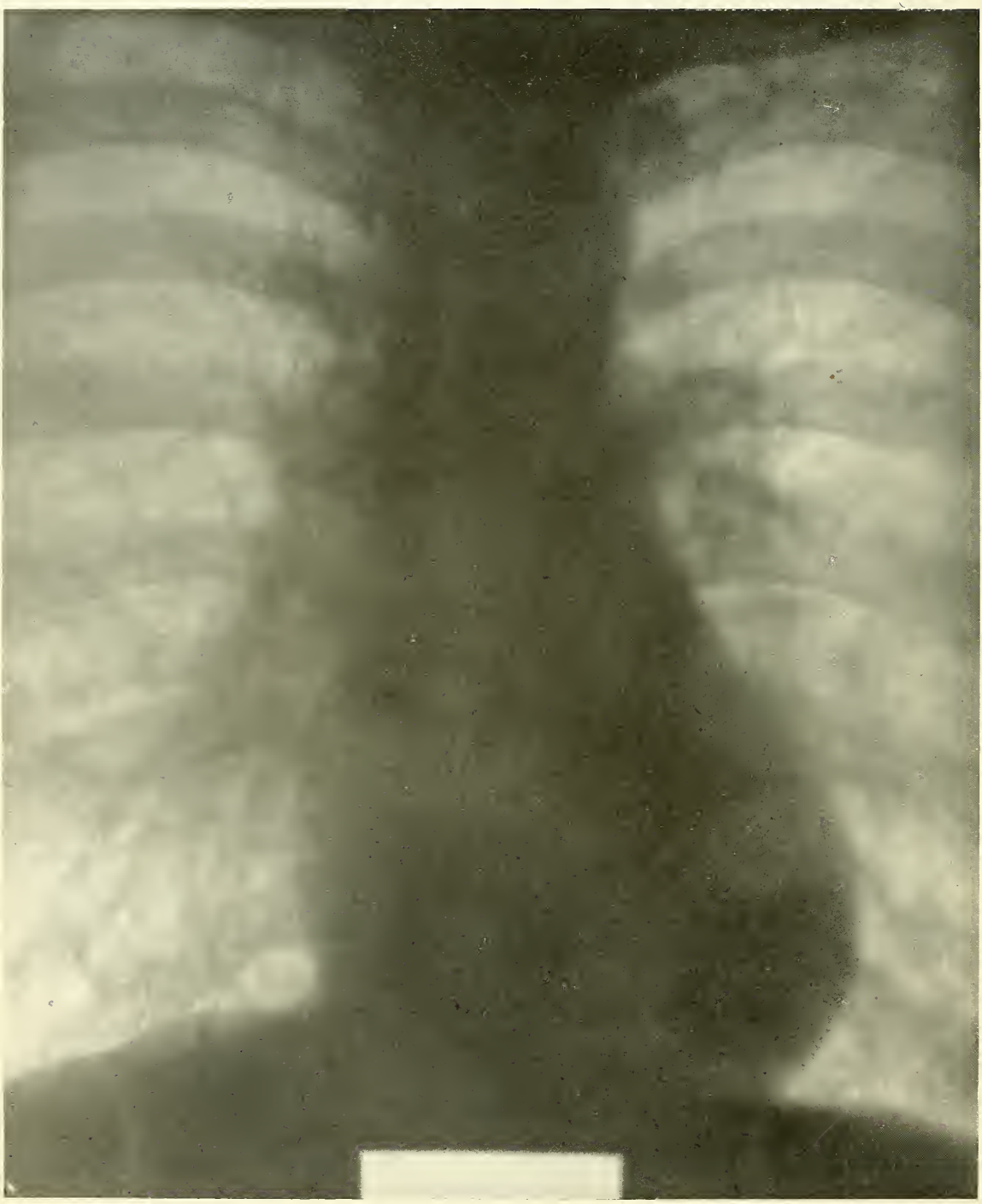

Fig 63. Bronchial dilatation of the right lower lobe (compare with Fig. 64).

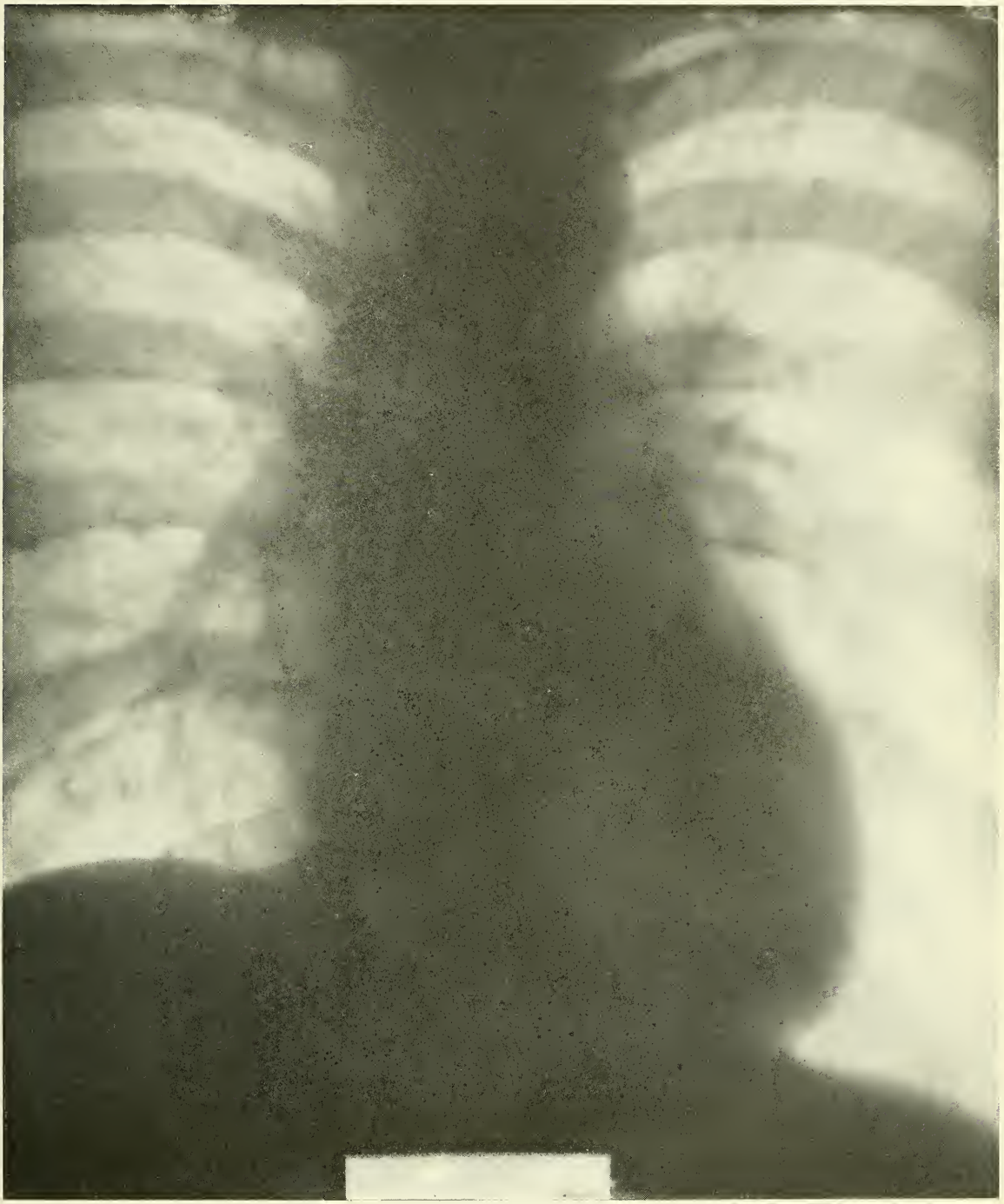

Fig. 64. Same case after section of the right phrenic nerve and consequent paralysis of the right half of the diaphragm.

to paralyse the diaphragm by dividing the phrenic nerve in the neck as a preliminary to the treatment. On p. 22 it has been explained how the movement of the diaphragm is the responsible factor for the expansion of the lower lobe. Paralysis of this muscle will, therefore, limit the expansion of the basal part of the lung and so check the aspiration of pus and the extension of the disease into the lower lobe.

Technique.

The patient is anæsthetised with chloroform and the phrenic nerve on the affected side is divided in the neck (see p. 193). The arm on the same side is then drawn up above the head and an incision some 10 cm. long is made through the 3rd intercostal space across the anterior axillary line. The pectoralis major is drawn upwards out of the way, and the 3rd and 4th ribs are separated widely from each other. The exposed parietal pleura is stripped with the fingers from off the chest wall over the upper part of the lung. During this process it is advisable that the anæsthesia should be as light as possible, so that the patient can cough up any secretion which is forced out into his trachea. As the pleura is freed from its attachments, the apex of the lung collapses and draws the membrane with it. The mass of fat or fibroma is inserted into the space which is thus left between the parietal pleura and the chest wall. The divided intercostal muscles are sutured and the wound closed.

Paralysis of the Diaphragm by Section of the Phrenic Nerve.

Section of the phrenic nerve in the neck will, in the majority of cases, produce complete paralysis of the diaphragm on the same side. Occasionally, some, or even all, of the motor fibres are derived from the nerve to the subclavius muscle and join the phrenic below the point of section. When one half of the muscle is completely paralysed, the arch of the diaphragm rises into the thorax and occupies a position about an inch higher than normal during quiet inspiration and higher still during deep inspiration. (Figs. 63 and 64.)

The effect of such paralysis is (1) to produce a diminution in size of the lower part of the pleural cavity and consequently of the volume of the lower lobe ; (2) great diminution or complete absence of expansion of the lower lobe.

The advantages gained by these changes make the simple operation of division of the phrenic nerve a valuable accessory to treatment—

A. *As a prophylactic measure.*

(1) Against the extension of the disease from the upper to the lower lobe, as already pointed out in the preceding section.

(2) Against the development of bronchial dilatation and distortion in the lower lobe, the diminution in volume of the base of the lung compensating for the mechanical disabilities which are the outcome of fibrosis in that region.

B. *As a method of treatment of symptoms.*

It has previously been pointed out that adhesions between the base of the lung and the diaphragm may be the cause of a cough so constant and irritating that the patient prefers to lie in bed. Section of the phrenic nerve will abolish this reflex and give the patient very great relief not only from the cough, but sometimes also from the dyspnœa which is associated with the fibrosis of the lung, the distortion of the diaphragm and the loss of the synchronous movements of the two structures.

C. *As a method of treatment of disease.*

(1) In the rare cases in which tuberculosis invades primarily or mainly the lower part of the lung, the immobility or the diminution of movement and the partial collapse of the base of the organ produced by paralysis of the diaphragm will have a beneficial influence in placing the granulomata under conditions favourable to their success and in overcoming the local mechanical disabilities.

(2) The success of nitrogen displacement is at times interfered with by the fusion of the diaphragmatic surfaces of the visceral and parietal layers of the pleura. This fusion will limit the degree of collapse obtainable partly by reason of the fixation of the lung and partly because of the symptoms which result from the drag on the muscle by the displaced lung. Paralysis of the diaphragm will allow of a considerable further collapse of the lung and will prevent the development of the symptoms due to the pull on that muscle.

(3) In certain cases of chronic fibroid phthisis in which there are few, if any, signs of activity, but in which the symptoms due to distortion and dilatation of the bronchi are progressive, the change in the mechanical conditions which will result from paralysis of the diaphragm will check the increase in these symptoms and may even diminish them.

Technique.

This operation is easily done under local analgesia, the tissues being infiltrated with 2 per cent. novocaine and adrenalin one hour previously, and a third of a grain of morphia given as well in nervous patients fifteen minutes before the operation. The nerve is found by making an incision $1\frac{1}{2}$ inches in length along the outer border of the lower end of the sterno-mastoid muscle. This muscle is retracted inwards, the scalenus anticus is

exposed and the nerve will be found running along the anterior surface of the muscle near its inner margin. The nerve is cut with scissors, the divided ends being left in apposition and the cutaneous wound is closed.

There are, therefore, at our disposal four different methods of surgical intervention in pulmonary tuberculosis. All of these are directed against those special adverse factors in the disease (*e.g.*, the constant movement, the mechanical disabilities, the secondary infection, hæmoptysis) for the improvement of which therapeutic, hygienic measures and graduated exercises are of such temporary and limited value. Medical and surgical treatment are both equally important, but each has its own sphere of influence and neither interferes with, overlaps or limits the functions of the other, the two dovetailing together with remarkable exactitude. The advantages gained by treatment by the combined forces have completely revolutionised the prognosis of the disease.

Chapter IX.

BRONCHIECTASIS.

Bronchiectasis is never a primary disease produced by some special organism, as is pneumonia or phthisis, and cannot originate in a healthy lung. It is the name given to a group of changes affecting principally the walls and lumen of the bronchial tubes which develops as a result of abnormal mechanical conditions in the lung parenchyma or of mechanical interference with the free passage of air. Bronchiectasis is, therefore, a late manifestation of some pre-existing disease or abnormality, and according as it develops directly in the course of the disease or at an appreciable interval after the subsidence of the primary lesion, may be regarded as a complication or as a sequela.

Bronchiectasis is always acquired. The so-called congenital form is not present at birth. It is the result of the absence of expansion (atelectasis) of the lung, but the symptoms of the dilatation of the bronchi develop so early in life that it is not only referred to as congenital bronchiectasis but is often erroneously regarded as such.

The two principal variations from the normal which are responsible for bronchial dilatation and distortion are : (1) The traction force of abnormal fibrous tissue formation in the lung parenchyma, and (2) interference with the free passage of air in the bronchi. The mechanical disabilities which are the direct consequence of fibrosis in the lung have already been discussed in the chapter on pulmonary tuberculosis (pp. 147 *et seq.*), but it may be briefly repeated that fibrous tissue, when it is the result of pathological changes, is in a constant state of contraction, and that the force of this contraction is exerted equally in all directions throughout the lung and indirectly also on the mediastinum, on the diaphragm and on the thorax. The direct effect of the constant pull on the walls of the bronchi is a progressive distortion and dilatation of the lumen of the bronchial tubes. A second and very important result of this fibrous tissue contraction is that it causes irregular constrictions which affect both the lung parenchyma and the bronchi, and causes, therefore, interference with the free entry and exit of air through the bronchus into the area of lung which it serves. Fibrosis is one only of several factors which may cause obstruction to a bronchus,

The influence on the bronchi of any interference with the free passage of air, from whatever cause, is described by Sauerbruch* as follows :— "The difficulty (in the to and fro movement of air caused by the obstruction) is of much less importance during inspiration than during expiration. The stenosis is easily overcome during the powerful active inspiration. The air can thus enter. Normal passive expiration is not, however, sufficient to drive out the air. More air is pumped into the affected bronchi during inspiration than is expelled during expiration. Forcible expiration, as in coughing, shouting and singing, causes thus a very considerable rise of pressure, which, when it is constantly repeated, readily produces a widening of the affected bronchi." This difficulty in driving out the air becomes greater when the stenosis is increased by the accumulation of secretions.

Bronchiectasis may develop :—

(1) As a sequela of :—

(*a*) An acute specific infection of the lung such as a pneumonia (particularly when due to influenza) which runs an atypical course failing to undergo complete resolution.

(*b*) An acute infection of the lung such as occurs as a result of gunshot wounds.

(*c*) Acute irritation of the bronchi, such as is produced by exposure to shell gas.

(*d*) Acute partial or total obstruction of the bronchus by a foreign body within the lumen.

(2) As a complication of :—

(*a*) Atelectasis of the lung—"congenital bronchiectasis."

(*b*) Any chronic infection of the lung such as phthisis and chronic abscess.

(*c*) Pressure on a bronchus from without, as a result of aneurism, enlargement of bronchial or mediastinal glands, chronic interstitial mediastinitis or foreign body (shell fragment or bullet).

(*d*) Chronic obstruction of the lumen of a bronchus due to intra-bronchial neoplasm.

Whether bronchiectasis develops as a sequela of some acute disease or as a complication of a chronic infection of the lung, or of obstruction of the air passage, there is practically no difference in the character of the dilatation and distortion of the bronchial tubes, nor in the nature of the symptoms. Such difference as there is, is to be found rather in the method of onset in the clinical picture. It is only when bronchiectasis develops as a sequela that there may be a definite latent period after the subsidence of the acute

* "Die Bronchiectasen." IIIe Congress de la Societe Internationale de Chirurgie. 1911.

symptoms of the primary disease and before the characteristic clinical picture due to the changes in the bronchi manifests itself. It is the existence of this definite and sometimes considerable interval of time, during which the patient, though never really well, does not manifest any apparently serious group of symptoms, which is responsible for the misconception which sometimes exists that bronchiectasis may be a disease *sui generis*.

A short account of the changes which take place in the lung will help to make clear the difference in the development of the clinical picture of bronchiectasis when it occurs as a sequela or as a complication. In a pneumonia which has failed to undergo complete resolution, three stages in the pathological processes can be recognised. The first stage is that of the acute pneumonic consolidation and toxæmia. This subsides gradually, but as the pneumonia undergoes incomplete resolution only, fibrous tissue formation occurs and becomes in some cases steadily progressive.* The second stage is that of fibrous tissue formation and contraction, which, when untreated, persists and continues until the death of the patient. The third stage is reached when, as a result of the mechanical changes, the bronchial tubes become deformed and dilated. Corresponding with these three stages there are three phases in the clinical picture. In the first the symptoms are acute and consist mainly of those indicative of the absorption of virulent toxins, together with cough and expectoration. The transition from the first to the second phase is fairly abrupt and is defined by the cessation of the acute manifestations rather than by the development of new ones. The symptoms of the second stage are few and, apart from the shortness of breath and possibly a slight persistent cough, are rarely definitive. The shortness of breath is not severe and is often regarded as an expression of absence of recovery of tone and of the general health rather than as an indication of an active process. The transition from the second to the third phase is, as a rule, so gradual that it is impossible to say at what precise period in the history of the case the phenomena due to bronchial dilatation have become superimposed on those due to fibrosis.

Occasionally an acute infection of the nature of a cold or of influenza occurring during the second phase is responsible for an abrupt and persistent change in the symptoms. As the acute manifestations of the fresh infection subside, it is found that certain symptoms and signs persist and that the clinical picture characteristic of bronchiectasis has become established. Under these circumstances the patient is apt to disregard the second phase, to lose sight of the importance of the primary disease and to date the origin of his present condition from the second acute infection.

Very similar are the changes observed when bronchiectasis follows on the aspiration of a foreign body into a bronchus, causing partial or complete obstruction of it. In these cases, however, owing to the continuous

* The progressive character of the fibrosis is very probably due to the persistence in the lungs of a mild chronic infection.

intrabronchial irritation, the symptoms of the first phase may not disappear, but gradually changing in type from acute to chronic, they continue as such until the development of the manifestations due to the establishment of the bronchiectasis, the period of the chronic symptoms representing the second or transitional phase.

The evidence that has so far become available indicates that in some of the cases which have exhibited very severe pulmonary symptoms as a result of exposure to shell or drift gas, there develops an interstitial fibrosis in the neighbourhood of the main bronchi. If the fibrosis is progressive in character, the adverse influence of this change on the walls of the bronchi becomes apparent. Time has not as yet been sufficient to show how serious the mechanical disabilities which may follow as a sequela of the irritation due to the inhalation of poison gases may be. It is certain that in many cases the fibrosis has been sufficiently intense to produce a very considerable, and possibly permanent, interference with the freedom of respiration, and that in a few, indubitable evidence of bronchial dilatation, especially of the larger tubes, has appeared.

The symptoms of bronchiectasis, when it occurs as a complication of some chronic pulmonary lesion, are often overshadowed by the symptoms of the primary disease. The development of the pathological changes in the bronchi may cause no alteration in the character of the clinical picture, but a very gradual aggravation of it only. It is consequently extremely difficult to estimate, at any given time, to what extent the primary chronic infection or the complication of it is responsible for the symptoms and signs. A radiogram may, however, be of great value in revealing the presence and extent of the bronchial dilatation. It is of considerable importance to obtain this evidence, as the presence of such change must be taken fully into account when considering the treatment and the prognosis of the disease.

Bronchiectasis may be unilateral or bilateral ; it may involve the whole of one lung, one lobe or a part of a lobe only. Limitation of the disease to the terminal main bronchus of the lower lobe is suggestive of a foreign body as the original cause. Implication of the greater part of the bronchi of one lung, together with extensive interstitial fibrosis (the condition which is usually termed *chronic interstitial pneumonia*), is most often the result of some previous acute infection, due particularly to the influenza bacillus or the pneumococcus.

The changes in the bronchi consist at first of swelling of the mucous membrane followed later by desquamation of the lining cells, by hypertrophy of all the coats of the walls and by dilatation of the lumen. Still later atrophic changes take place in localised areas ; these are separated from each other by areas of hypertrophy. As a result of this atrophy the walls become thinned and yield gradually to the pressure of air in the lumen of the tube. The change in the shape of the bronchus depends therefore on the extent of the areas of atrophy. If these involve the whole circumference of the

lumen, a cylindrical or fusiform enlargement of the bronchus will take place and a series of such dilatations may occur along the course of one or more bronchi. Occasionally the atrophic process is very pronounced, but is limited to a part only of the circumference of the tube ; this part of the wall stretches and eventually forms a cavity projecting into the lung parenchyma and communicating with the bronchus by a lateral opening.

These changes in the bronchi interfere with the normal removal of the secretions of the mucous membrane, and secondary infection is the inevitable result. The severity of the infective processes is proportionate to the extent to which drainage of the dilated bronchi, and especially of the cavities, is interfered with. When the secondary infection is acute, the surrounding lung shows pneumonic consolidation. Very occasionally this consolidation is not limited to a zone of lung tissue immediately adjacent to the cavity or bronchus, but affects the whole lobe or even the whole lung. Rarely the action of the pyogenic organisms may result in ulceration of the thin atrophic wall of the bronchus and produce a chronic interstitial abscess.

The opening into the cavity leading out of a dilated bronchus, or even the lumen itself of one of the bronchioles, may become partially, or for a time totally, obliterated. This is followed by an increase in the virulence of the organisms and a serious increase in the absorption of the toxic products. The obstruction, which is produced by a plug of mucus, seldom lasts more than five to seven days, and usually yields at the time when the symptoms are becoming so serious as to cause considerable anxiety. The re-opening of the channel of communication is probably effected by the disintegration of the plug by the digestive action of the organisms. When the obstruction obliterates the lumen of a bronchiole, there is a change of the nature of massive collapse in the area of the lung supplied by that bronchiole.

The presence of these inflammatory changes in the lung is not infrequently responsible for local inflammation of the pleural membranes. An effusion is extremely rare, but local thickening of the membranes and adhesions are common.

When the disease is limited to one side or to one lobe and has been present for some time, there are always certain changes in the larger bronchi of the opposite side also, specially in those supplying the lower lobe. These changes are due to a congestion and hypertrophy of the mucous membrane as a result of the chronic irritation caused by the overflow of secretions from the affected side.

The combination of bronchiectasis with abscess formation has already been alluded to in Chapter VII, and it was stated there that the abscess might be due to an interstitial pulmonary abscess which has developed as the result of ulceration of a bronchus, or that the chronic abscess may be the primary condition and the bronchiectasis a complication of it.

Fig. 65. Bronchiectasis involving the right lower and middle lobes, and, to a less extent, the upper lobe on the same side. This radiogram was taken three weeks after that shown in Fig. 66.

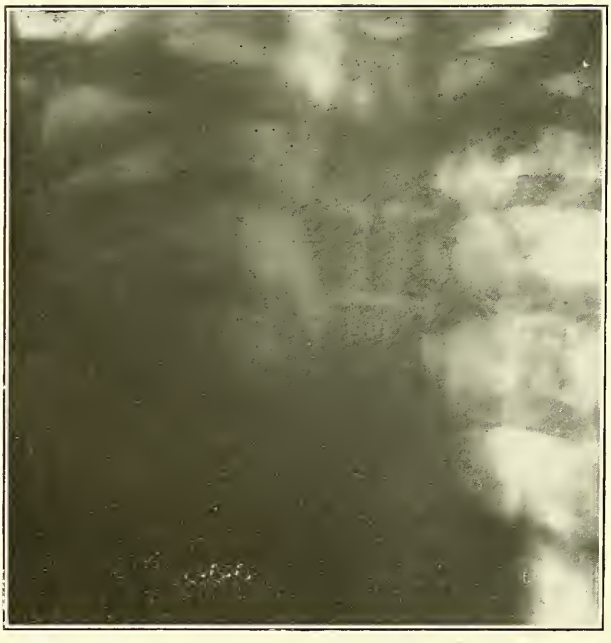

Fig. 66. Same case as shown in Fig. 65, but taken three weeks earlier. Massive collapse of the lung. *Radiograms by Dr. Kincaid.*

The bacteria found in the expectoration vary greatly. In some cases cultures from the sputum show a mixture of pyogenic, non-pyogenic and saprophytic organisms, while in others, and these are less common, an almost pure growth may be obtained. It will usually be found, however, that even when there are several varieties present, one of these predominates over all others. The putrefactive changes associated with the attacks of retention of the secretions in a dilated cavity or cavities, or in an interstitial abscess, are due to the action of the saprophytic organisms.

SYMPTOMS AND SIGNS.

Bronchiectasis is a chronic condition, developing insidiously, progressing steadily, but subject to exacerbations due to imperfect drainage. The characteristic symptoms are the periodic cough and expectoration; the former is to a great extent dependent on the latter. The dilated bronchi acquire a tolerance of the secretions; changes in posture or overflow of secretions by accumulation cause irritation of the less tolerant mucous membrane which results in coughing and the expectoration of a considerable quantity of the collected sputum. Pain is often present when there is secondary inflammation of the pleura; both the pain, and the friction rub when heard, are localised, usually to quite a small area. Thickening of the membrane and adhesions may occur, however, without causing symptoms. The character of the sputum closely resembles that of chronic abscess and has already been described on p. 126. Elastic fibres are not found except in those rare cases where, as a result of ulceration of the bronchus, an interstitial abscess has formed.

In all cases of long standing chronic suppuration of the lung and pleura, the condition known as hypertrophic pulmonary osteo-arthropathy is found. The changes are most noticeable in the fingers, the terminal phalanges of which, including the nails, are greatly enlarged (clubbed fingers). The hands in general and the feet, and at times even the nose, are similarly affected. This disorder is as a rule more pronounced in bronchiectasis than in any other disease.

The clinical picture may be that of chronic bronchitis, of chronic interstitial pneumonia, of fibroid phthisis, of chronic abscess, or of multiple cavitation. The physical signs may vary also in the same individual at different times according to whether the cavities are full or comparatively empty of secretions. The greatest variability is noticed, however, in those cases in which there is a chronic cavity associated with the bronchiectasis; the cavity being not necessarily in the lung parenchyma. During the intervals between the attacks of retention, the patient is moderately well, there is a certain amount of cough and the expectoration is anything from 30 to 100 c.c. of sputum a day; the expectoration is mainly in the morning and evening. When an attack of retention comes on, the cough diminishes,

the purulent sputum is, to a great extent, replaced by a mucoid expectoration, the temperature rises a little higher each day and the patient feels progressively more ill and the breath becomes increasingly offensive. Quite suddenly communication is re-established with the bronchus, a large amount of offensive sputum is expectorated, the temperature drops and the patient returns to his previous state of health. This cycle of changes may occur with the greatest regularity, in some patients at short and in others at long intervals of time.

RADIOLOGY.

The radiological appearances of bronchiectasis, when it occurs as a sequela, are by no means uniform. Four different types can be described.

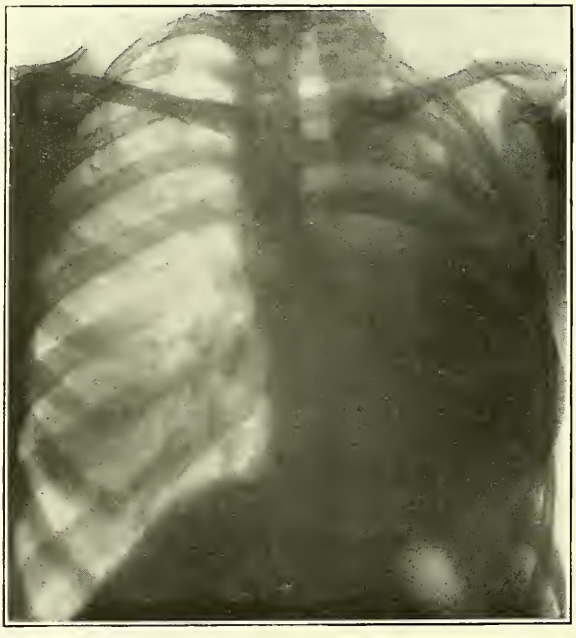

Fig. 67. Old standing bronchiectasis of the left lung (chronic interstitial pneumonic type). Note the displacement of the heart and of the trachea to the affected side; the distortion of the diaphragm by adhesions and the secondary scoliosis.

The first three of these are so characteristic that the condition, if advanced, can be diagnosed from the radiogram alone. In the fourth type, that in which bronchiectasis is complicated by a cavity, the X-ray appearance is indistinguishable from that which is obtained in a case of primary abscess of the lung complicated by bronchial dilatation. Practically every case of bronchiectasis will be found to conform with one or other of these groups.

1. *The chronic interstitial pneumonic type* (Fig. 67). There is considerable diminution of the lung volume and a corresponding traction on

and displacement of the surrounding structures ; the displacement of the trachea, of the heart and of the mediastinum is a striking feature of the radiogram. The affected part of the lung has a homogeneous ground glass appearance ; the shadow is usually most intense at the base, but is rarely as dense as a pleural effusion, or as the shadows given by the heart and the diaphragm. The radiological picture is closely similar to that found in

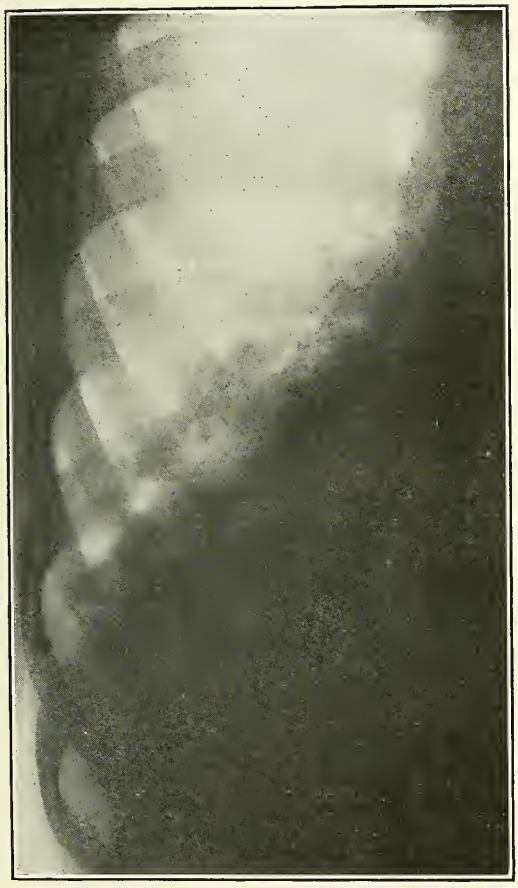

Fig. 68.

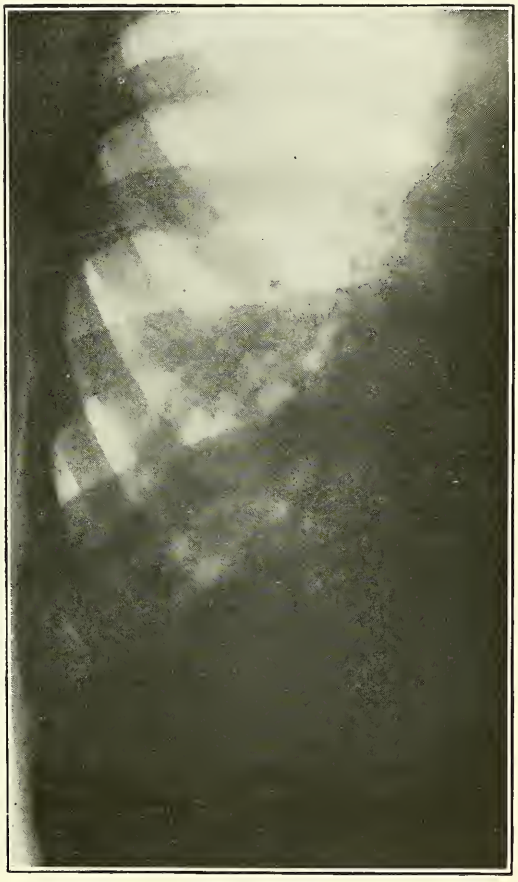

Fig. 69.

Fig. 68. Bronchiectasis of the right lower lobe. The mesial bronchi are the most affected.

Fig. 69. The same case. Skiagram taken fifteen minutes after that shown in Fig. 68. In that interval the patient had been inverted and had coughed up four ounces of sputum.

some cases of long standing chronic phthisis, a condition in which bronchial dilatation is always present as a complication of the primary disease (*cf.* Fig. 67 with Figs. 48 and 49). In these cases, however, the uniformity of the ground glass appearance is not so pronounced and in some parts of the

lung, usually in the region of the upper lobe, some detail of the lung tissue or of the disease can be made out. The condition also of the hilum of the opposite lung is of assistance in the differentiation (see p. 205).

In some cases the homogeneity of the shadow may be due to an acute pneumonic change or to massive collapse supervening on the bronchial lesion. This is rare, but is well seen in Figs. 65 and 66. The latter of these was taken during the period of complication by massive collapse, and the former three weeks later after the subsidence of it.

2. *The multiple dilated cavity type.* Figs. 68 and 69 show clearly the difference in the shadows obtained, according to whether the cavities are full or comparatively empty. This condition may be unilobar, as in the illustration, it may be symmetrically bilateral or involve a series of lobes to a greater or less extent.

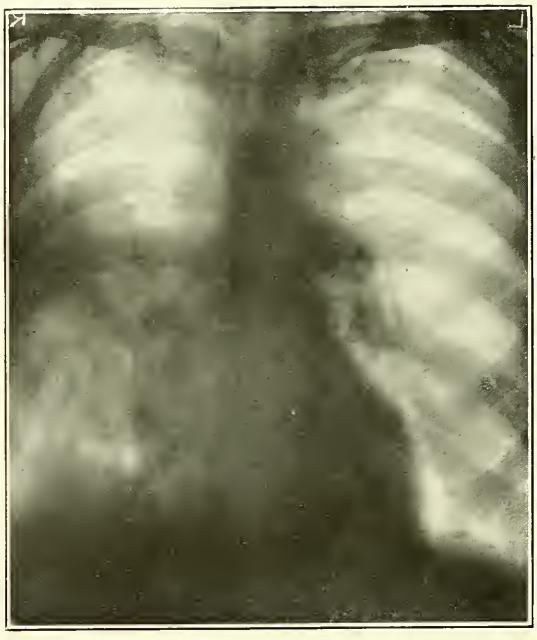

Fig. 70. Chronic abscess with secondary bronchiectasis.

3. *Bronchiectasis limited to the inner part of the lower lobe.* This is a not uncommon situation, and when present on the left side the shadow in the lung due to the pathological changes is often difficult to see, as it is in a great measure obscured by that of the heart. Ventro-dorsal and semi-lateral radiograms must always be taken. In this and the preceding type, the localisation of the lesion suggests that the disease is a sequela of a simple basal pneumonia or of a foreign body in the bronchus. A very noticeable

feature of, particularly, the first and the third of these types is the very great increase in obliquity and approximation of the ribs. A marked change in the position of the ribs on one side of the chest associated with comparatively little evidence of disease in the outer and easily visible area of the lobe may be regarded as definite evidence of a bronchiectasis which is limited to the inner part of the lower lobe.

4. *The chronic abscess type* (Fig. 70). There is a considerable amount of thickening of the hilum shadow, and beyond in the substance of the lung there is an area of more uniform density. The size of this area, the definition of its outline and the density of the shadow may vary very greatly from time to time in the same patient when the case is one in which there are periodic attacks of complete occlusion of the mouth of the cavity and consequently of complete retention of the secretions.

In practically all cases in which there has been a unilateral bronchiectasis of some duration, the hilum on the opposite side will give a considerable shadow owing to the hypertrophy of the main bronchi, specially of the lower lobe, caused by the irritation of the purulent secretions.

TREATMENT.

In order to understand the principles underlying the more radical methods of treating bronchiectasis, it is necessary to recapitulate certain facts. The type of bronchiectasis under immediate consideration is not that which occurs as a complication of a chronic disease such as phthisis, but that which develops as a sequela of a previous acute infection, of congenital atelectasis, or of obstruction of a bronchus by a foreign body. The essential feature of the morbid anatomy, other than the dilatation of the tubes, is the fibrosis of the interstitial tissues and the interference with the free movement of air through the bronchi. The symptoms which cause the patient the most distress are due to retention of secretions and to infection, and these are entirely secondary and a necessary corollary to the above changes. It is these conditions also which are responsible for the deterioration of the patient's health and which endanger his life. There is, moreover, a vicious circle established—the fibrosis leading to the changes in the bronchi which make possible the retention of secretions, whilst the infection in the tubes causes irritation of the adjacent parenchyma and an increase of the fibrosis.

Medical measures can, in many cases, when the retention is due to postural rather than to mechanical difficulties, diminish the virulence of the infection and consequently the amount of the secretion. The symptoms and the patient's general condition are thereby improved. Such improvement cannot, however, be more than of a temporary nature as no improvement of the mechanical conditions has been produced ; nor can it occur at all if there is retention due to actual mechanical interference with the escape of the secretions. It is obvious, therefore, that in order to obtain a permanent improvement, some method of treatment must be adopted which arrests

the progressive fibrosis and which directly or indirectly produces approximation of the walls of the secondary and tertiary bronchi and of the air passages beyond. Such approximation of the walls will obliterate the cavities, will prevent the retention of secretions and will thereby greatly diminish the infective processes.* The measures which are necessary for obtaining the obliteration of the dilated spaces must at the same time ensure that no further traction on the walls of the bronchi by the fibrous tissue will take place.

The above changes which it is essential should be effected in order to obtain a more permanent relief from the symptoms of bronchiectasis, and to enable the patient to resume, in some measure at any rate, his former life without being a burden to himself and an offence to his neighbours, can be brought about by :—

1. Collapse of the lung only.
2. Collapse of the lung together with collapse of the chest wall—rib mobilisation.
3. Local collapse of the chest wall and paralysis of the diaphragm.
4. Ligature of the branch of the pulmonary artery supplying the lower lobe.

Every case of unilateral bronchiectasis is suitable for surgical treatment, but only very few of those cases in which the disease affects both sides. It has already been pointed out that in most cases of unilateral bronchiectasis of long duration, there is some hypertrophy and possibly dilatation of the main bronchus, and sometimes of the secondary bronchi of the opposite lower lobe also. This is no contra-indication to treatment. Cases in which there is definite involvement of both lower lobes and of no other part of the lung can also be dealt with surgically, but the prognosis is less hopeful.

TREATMENT BY COLLAPSE OF THE LUNG.

(*Nitrogen displacement.*)

In a case in which there are no pleural adhesions it is possible by injecting nitrogen into the pleural cavity and changing the negative intrapleural pressure to a positive one to obtain complete collapse of the lung, and consequently collapse and approximation of the dilated bronchi. This prevents the accumulation and retention of secretions and leads therefore to a decrease in the virulence of the organisms and to a striking diminution in the absorption of the toxins. As a natural corollary of these changes the great majority of the symptoms disappear and there is a rapid improvement in the patient's general condition.

This satisfactory result lasts so long only as a positive pressure on the surface of the lung is maintained by repeated further injections of gas into

* It is quite rare for a dilatation of a primary bronchus to be associated with obstruction of the mouth of it. If there is no obstruction there is free drainage, and there is therefore no necessity, though it would be advantageous, to produce approximation of the walls.

the pleural cavity ; since if the gas, as it is absorbed, is not replaced, the intrapleural pressure diminishes, the lung begins to re-expand and the lumen of the irregularly dilated bronchi to re-open ; the infection which has been in abeyance resumes its activities and the symptoms due to the accumulation of secretions and to the absorption of toxins return. It is necessary, therefore, in order to maintain the improvement, to keep the surface of the collapsed lung exposed to a continuous positive pressure. This is possible only by giving a fresh injection of nitrogen at least once a fortnight during the first six months of the treatment, and at slightly longer intervals during the later periods. This treatment, moreover, must be persisted with almost indefinitely, since in a bronchiectatic lung there are very few permanent advantages gained as compared with a tuberculous lung. In the latter the immobilisation has placed the granulomata under conditions definitely favourable for controlling the organisms, and the collapse of the lung has allowed the fibrous tissue to become transformed into healthy scar tissue and the cavities to become obliterated by the approximation and healing of their walls. In a bronchiectatic lung, the dilatation and distortion of the bronchi are permanent changes ; collapse of the lung will enable the fibrous tissue to undergo the contraction necessary for the formation of a healthy scar and may lead to the obliteration of an interstitial abscess, but it will not result in the permanent obliteration of cavities lined by mucous membrane. When, therefore, the lung is allowed to re-expand, the cavities will open up also.

It is evident that, whilst nitrogen displacement cannot be regarded as a satisfactory permanent method of treatment for bronchiectasis, yet as a temporary measure and as a preliminary to a more radical operation, it is often invaluable. The operation of rib mobilisation which is described below, and which is advocated as the most satisfactory procedure for most cases of this disease, is a severe one, and makes a considerable call on the reserves of the patient. For this reason it is of the utmost importance that every possible endeavour should be made to improve the general health of the patient by every means within one's power. Among those available, in cases in which there are no pleural adhesions, the preliminary collapse of the lung by nitrogen displacement, and the consequent abolition of the sapræmia, are of the greatest value ; the lung should be kept in a state of collapse by gas for one or two months before the more serious operation is undertaken.

The details of the technique of nitrogen displacement have been fully described in the last chapter, and there is only one difference in the production of an artificial pneumothorax in bronchiectasis from that in phthisis. In the latter condition, the sudden injection of a large quantity of nitrogen may cause a serious reaction, and must therefore always be avoided. In bronchiectasis, on the other hand, a reaction is rarely seen, and much larger quantities of gas can be run into the pleural cavity with safety on each occasion, so that complete displacement of the lung can be obtained by three, four or five injections.

TREATMENT BY COLLAPSE OF THE LUNG TOGETHER WITH THE CHEST WALL. (*Rib mobilisation.*)

This combined collapse of the whole lung and chest wall can be brought about either by the operation of rib mobilisation, or by that of total unilateral decostalisation. The former is so incomparably more satisfactory that this operation only will be discussed and described.

The operation of rib mobilisation is suitable for any case of unilateral bronchiectasis, whether uni- or multi-lobar, or whether the dilatation is uniform, is multiple and irregular, or consists of one large dilated cavity communicating with dilated bronchi. It is also the operation to be recommended for cases of chronic abscess associated with bronchial dilatation.

The mobilisation of the ribs is obtained by removing, in front, a part of the costal cartilages of all the ribs from the 1st down to and including the costal margin, and behind, a portion of all the ribs from the 1st to the 9th or 10th inclusive. The section of the 1st rib in front and behind and of the cartilaginous portion of the costal margin are both of them essential to the success of the operation. All the possible good results will be vitiated if the 1st rib is left untreated because the disease is in the lower lobe only, and most of them if the costal margin is left intact when collapse of the upper lobe only is desired. The operation is done in two stages, the posterior resection before the anterior one. The shorter the interval between the two, the more complete will be the collapse of the chest wall and of the lung, and the greater, therefore, the improvement in the patient's condition. Unless there is any contra-indication, such as feebleness of the patient or a reactionary fever, the interval between the two operations should not be more than ten days or a fortnight.

The changes that occur as a result of this rib mobilisation, and of the paralysis of the intercostal muscles by the injection of alcohol into the intercostal nerves, are that the whole chest wall on that side sinks directly inwards and downwards ; the ribs become approximated to the middle line and, at the same time, not only drop bodily downwards but their angle of inclination is greatly increased (bucket handle action), so that the cut ends of the free part are from 2 cm. to 6 cm., whilst the lateral convex margins are from 3 cm. to 9 cm., below their normal level. This results in a very pronounced diminution of the pleural space, as can be seen in Figs. 60, 61, 62 and 71.

The improvement which has been described as resulting from the collapse of the lung by nitrogen displacement are found in an even more striking degree after rib mobilisation. The more drastic operation produces the greater relief from the symptoms, because the collapse of the lung, though not quite so complete as at the end of a displacement by nitrogen, is permanent. The constant fluctuations in the size of the lung in the latter condition, which must necessarily be associated with variations in the size

of the bronchial cavities, and therefore also with the degree of infection, are absent after rib mobilisation. The chest wall during the ensuing months is still sufficiently mobile to yield to the further shrinkage of the lung, and consequently the force exerted by the fibrous tissue on the bronchi becomes negligible.

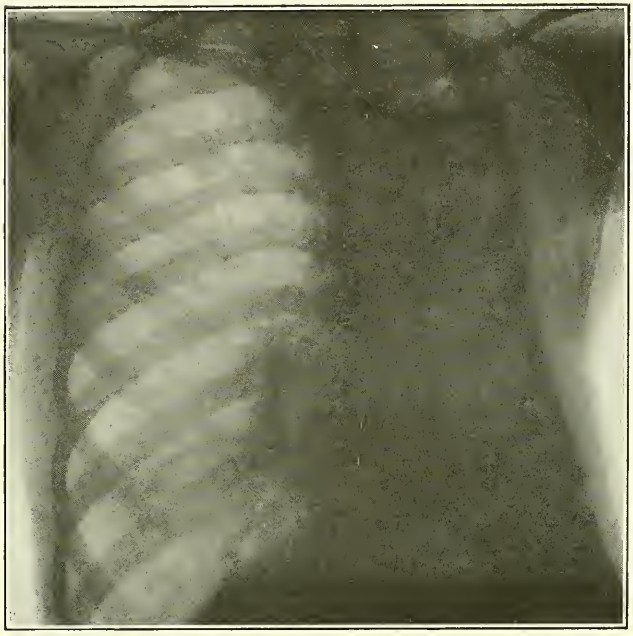

Fig. 71. Radiogram taken after the operation of rib mobilisation on a boy aged 11, suffering from bronchiectasis. Note the increase of the hilum shadow on the right side due to "overflow irritation."

The improvement which can be obtained by this operation depends to a great extent on the completeness with which the operation is done (and an incomplete operation is of comparatively little use), and on the shortness of the interval between the first and second stages. Bronchiectasis differs from phthisis in that the reaction after the first stage, particularly if a nitrogen displacement has been possible as a preliminary, is rarely so great as to prolong the interval which must elapse before the second operation can be done to more than three weeks or a month. An interval of longer duration allows of a sufficient degree of fibrosis and even of rib regeneration to take place in the posterior scar to interfere somewhat with the free collapse of the chest wall after the second stage.

The following brief description of a case of bronchiectasis, which had resisted medical treatment for many months, illustrates the improvement which may be expected after the operation of rib mobilisation.

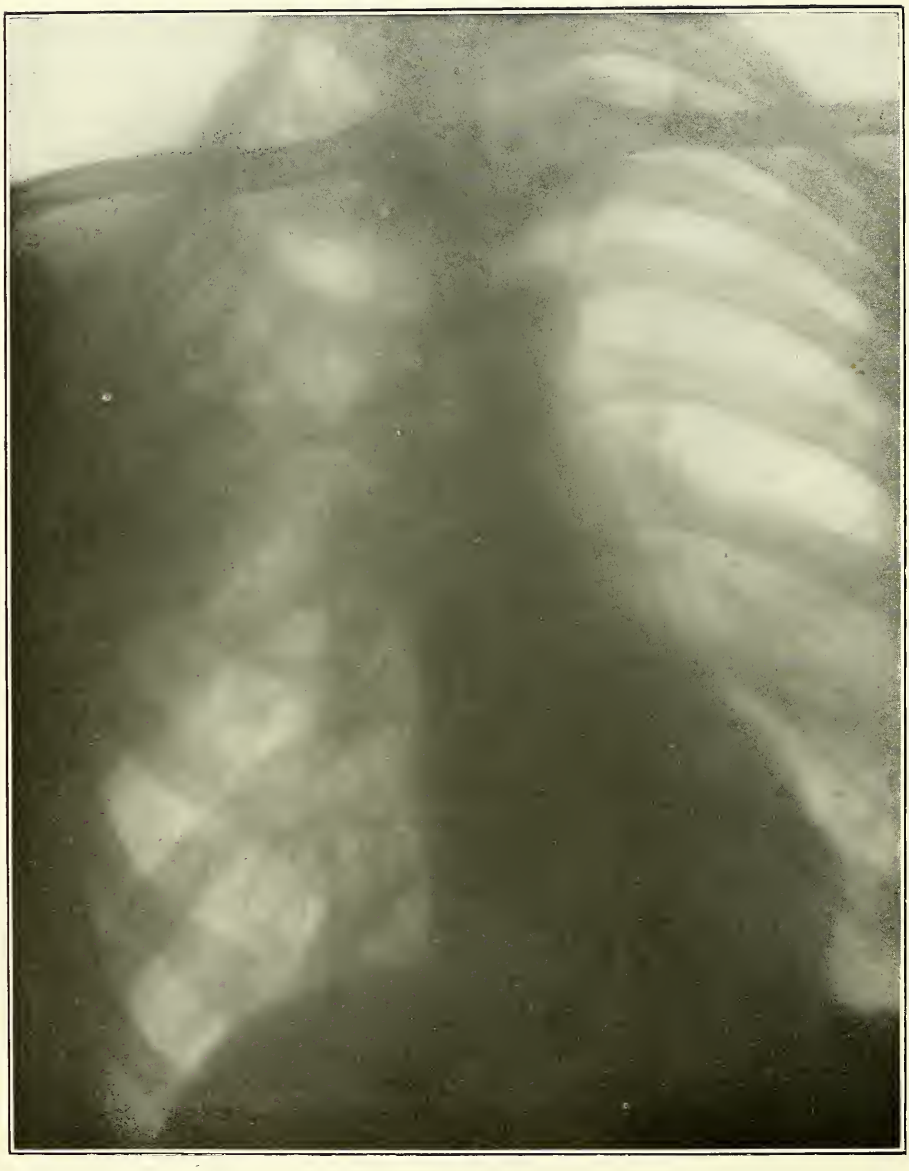

Fig. 72. Bronchiectasis and secondary abscess. Radiogram taken immediately after mobilisation of the upper ribs only. The costal margin was not divided: had this been done, a more complete collapse would have been obtained. (*Cf.* Fig. 61.)

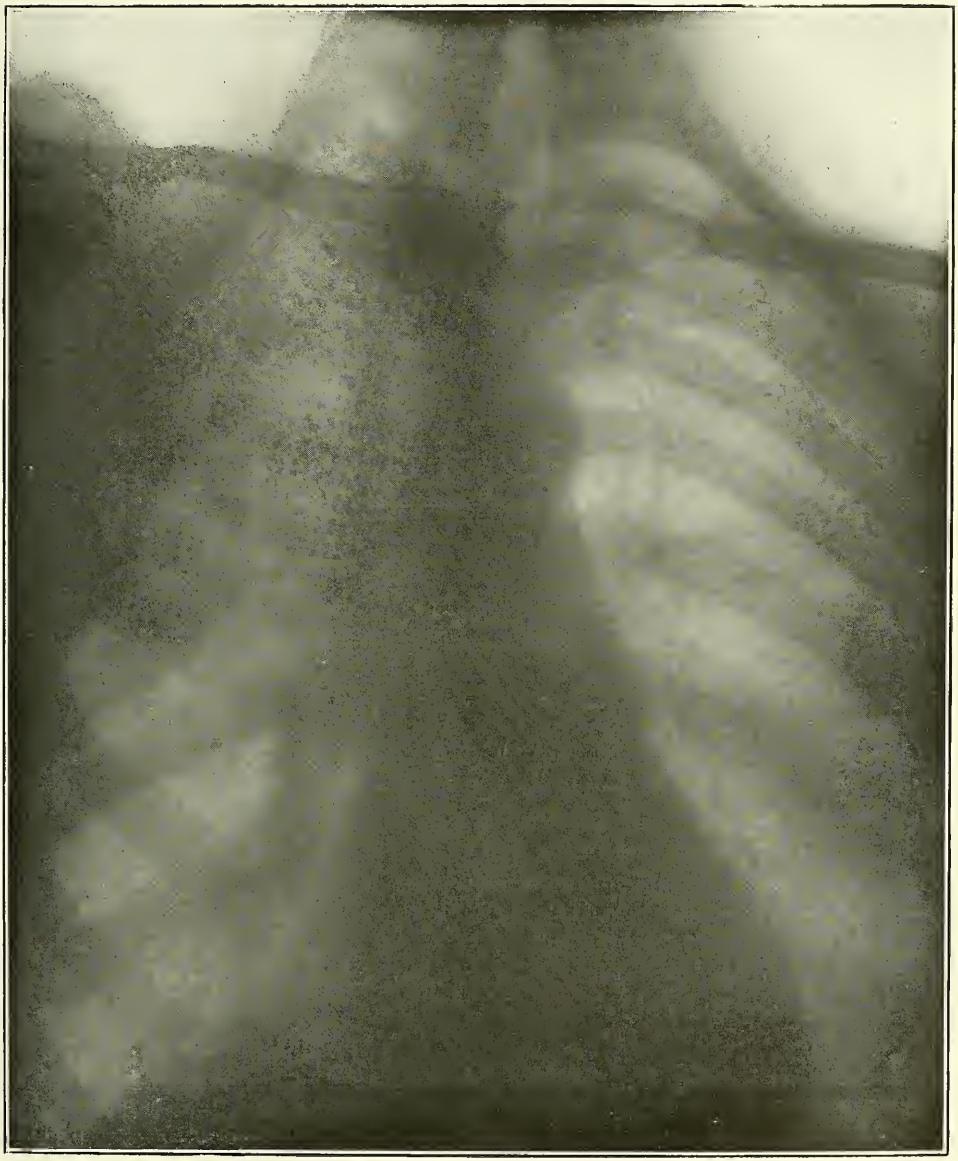

Fig. 73. Same case as Fig. 72. Radiogram taken two years later. Note the alteration in the shadow of the abscess and the increase in the collapse of the chest wall. The patient had had practically no symptoms during the preceding twelve months,

Following an attack of influenza in February, 1914, the patient, aged 43, developed bronchitis associated with a somewhat offensive expectoration. This condition persisted and there were occasional slight hæmorrhages. No tubercle bacilli were found in the sputum. In August of the same year, the cough and expectoration became worse and vaccines produced no improvement. In December there was a considerable hæmorrhage and a large quantity of offensive sputum was brought up. This was followed by a relief of symptoms until February, 1915, when all the old symptoms recurred together with a dry pleurisy on the right side. A radiogram showed extensive fibrotic changes in the right lung. From this time until August, despite vaccines, large doses of creosote and a fresh air life, the sputum increased, and the patient lost weight and became progressively weaker. In August, the signs and radiograms were conclusive of the diagnosis of a right-sided unilateral bronchiectasis involving the greater part of the right lung, except immediately at the apex and base. The expectoration and perspiration were very offensive. The general condition of the patient was fair and the cardiac condition good. The patient could not lie down owing to the violent attacks of coughing which immediately ensued.

On August 23rd, 1915, the first stage of rib mobilisation was done, portions of the 1st to the 9th rib being removed. The patient was anæsthetised with chloroform and the nerves injected with absolute alcohol. During the last forty minutes of the operation no anæsthetic was required. The second stage was done on September 9th under light chloroform anæsthesia only. The costal cartilages of all the upper ribs down to and including a portion of the costal margin were removed.

The patient improved rapidly during the next few months. By January, 1916, there was no cough and no sputum and a walk of three miles was possible. When seen in November, 1916, fifteen months after the operation, the patient stated that she felt absolutely well. She had increased in weight and could walk twelve miles without any fatigue ; there was very slight shortness of breath on going up hill. There were absolutely no symptoms, and no adventitious sounds could be detected.*

Age itself is rarely a contra-indication of the operation. I have done this rib mobilisation on a man over 70 and on a girl aged 7 years.

The Operation of Rib Mobilisation.

A certain amount of preliminary work must be done before the operation of rib mobilisation can be undertaken. Good radiograms showing the extent and the character of the changes are, of course, essential. The sputum should be examined so as to ascertain what organisms are present. An attempt should always be made to collapse the lung by nitrogen displacement. If this proves possible, the displacement once effected should be maintained

* This state of good health has been maintained until the present time (August, 1918).

for a period of at least three weeks, but in patients whose general condition is unsatisfactory, for at least six weeks before the operation. When the artificial pneumothorax is impossible, it is probably advantageous to give the patient an autogenous vaccine so as to reduce the virulence of the infection as much as possible. The patient is kept in bed and under observation for a week or ten days immediately prior to the operation, and longer if the toxic symptoms are at all pronounced. The dilated bronchi must be kept as empty as possible of secretions and for this purpose inversion of the patient is carried out twice a day, and an expectorant mixture is given. The importance of sugar before the operation has already been referred to on p. 130.

The operation can be done with the patient under the influence of morphia and regional anæsthetisation with novocaine ; this is not, however, recommended except in very exceptional circumstances. Chloroform must be given in preference to ether. When the intercostal spaces are exposed during the first stage (the posterior incision), each of the intercostal nerves should be injected with a few minims of absolute alcohol, as close to its exit from the intervertebral foramen as possible. The reasons for this are fourfold : it reduces the amount of the general anæsthetic necessary to keep the patient under, both during the first and second stages ; it diminishes the post-operative shock ; the patient suffers considerably less pain after the operation ; and the collapse of the chest wall is greater because the intercostal muscles are paralysed.

*Technique of the Operation of Rib Mobilisation.**

First stage.—The position of the patient on the table depends to some extent on whether he breathes more comfortably and coughs less in the sitting or recumbent posture. In the former case the patient is supported in a semi-recumbent posture, reclining somewhat on to the sound side while the arm on the affected side is held across the chest by an assistant. The surgeon, operating from behind, should have a clear view of the whole of the back of the affected side up to the vertebral border of the opposite scapula. When the recumbent posture is more satisfactory, or the patient is unable to tolerate any pressure on the sound side, he must be placed on the table lying well over on to the front of the chest on the affected side, the lower shoulder being drawn forwards as far as possible and the arm on the sound side supported. The former of these two methods is the more comfortable, both for the operator and for the anæsthetist. In either case the patient should be well supported so that he does not need constant re-adjustment during the operation.

A long vertical incision is made between the erector spinæ and the vertebral border of the scapula. Above, it begins on the back of the neck opposite the spine of the 6th cervical vertebra; below, it ends over the 10th

* Author's modification of Wilm's operation.

or 11th rib. All the muscles in the length of this incision overlying the ribs are divided and the posterior surface of the ribs and the intercostal spaces are exposed. The main vessels are ligatured, and oozing is checked by packing the wound with gauze, exposing during the rest of the operation only that section of the deeper parts on which the actual work is being done. An attempt is now made, using a hypodermic syringe and needle, to inject each of the intercostal nerves ; some 5 to 10 minims of absolute alcohol being introduced into the space between the pleura and the aponeurosis of the internal intercostal muscle immediately below the lower border of the rib. As the nerve is not exposed to view, an exact knowledge of its position, of course, is essential. During this and the ensuing part of the operation, the scapula must be drawn well over to the outer side ; the serrated retractor shown in Fig. 74 is recommended for this purpose. The exposed part of the

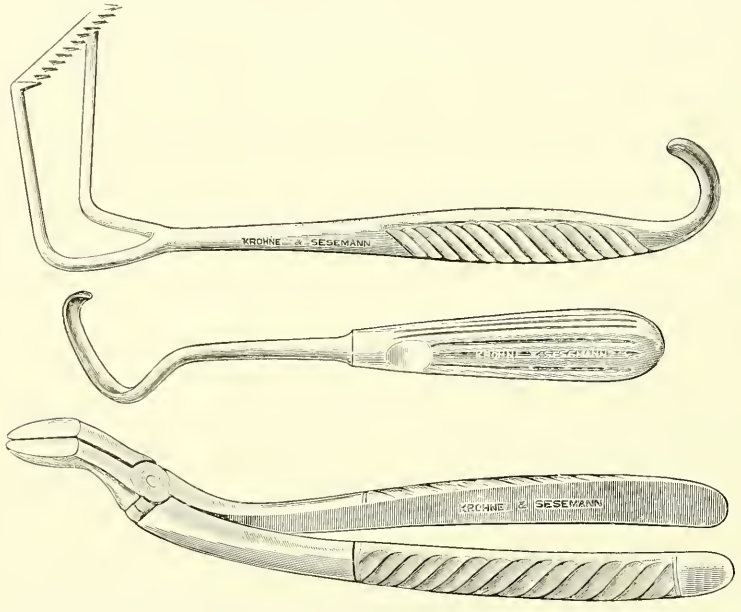

Fig. 74. Author's retractor, modification of Doyen's periosteal elevator, and bone forceps.

ribs, beginning with the 4th and 5th and working downwards to and including the 9th or 10th rib,* is now denuded of periosteum for a length of at least 8 cm. from the tubercle.† The denuded portion of the 4th rib is excised ; this affords easier access to the 3rd, which is in turn denuded of periosteum and cut away. The 2nd rib is similarly treated, but is less easy of access ; the first rib can now be dealt with. The length of bone which it is possible to remove decreases from the 4th to the 1st rib ; rarely more than 2 to 2·5 cm. of the uppermost rib can be excised through this incision.

* The 10th rib should be included whenever the disease extends to the base of the lower lobe.

† Owing to the depth of the wound, it is difficult to use the ordinary Doyen raspatory. The modification of this instrument (shown in Fig. 74) overcomes the difficulty.

There is considerable difficulty in exposing and resecting the posterior part of the 1st rib; this is due to the depth of the bone, to the shape of it (the surfaces being superior and inferior and the inner margin being practically invisible), and to the close relation of several important structures to the superior surface and to the inner margin. The anterior primary division of the 1st dorsal nerve passes over the inner border of the rib to join with that of the 7th cervical and form the lowest trunk of the brachial plexus. This trunk winds outwards round the anterior border of the scalenus medius, whilst immediately in front, between the nerve and the scalenus anticus muscle, is the subclavian artery. The cervical pleura and lung are internal to these structures. The part of the bone which is removed is that which extends from the tubercle to the insertion of the scalenus anticus. The scalenus medius is detached from the rib together with the periosteum, and the raspatory in doing this must never be allowed to slip off the bone. The detachment of the periosteum from the inner border is difficult and needs great care. When the bone is clear, a narrow copper spatula with the end bent over like a hook, must be passed along the inferior surface of the rib and the hook-like end brought round the inner margin on to the superior aspect. This spatula will guard the soft structures from injury when the bone is divided with the forceps. The bone forceps recommended for the division of the ribs is shown in Fig. 74; the handles are long, the blades are narrow and are sharpened to within 3 mm. only of the points, which are rounded and do not come into contact.

After the 1st rib has been divided, the denuded portions of bone of the lower ribs are excised. If the patient's condition is still good, it is advisable at this stage, but not essential, to remove some of the detached periosteum especially from the upper part of the incision. The divided muscles extending from the chest wall to the scapula are now sutured with catgut and the cutaneous wound closed without drainage. A large dressing is applied and secured with a many-tailed bandage. This should not be removed until the tenth day, when the stitches are taken out.

Second stage.—This second operation is not done until the temperature, which will have risen after the first stage, has again fallen and been at, or below, its former level for at least three days. In some cases, an exception to this must be made. The temperature may drop a degree or two after the initial reaction, but the daily maximum record is constantly somewhat higher than formerly.* In these circumstances, as delay may jeopardise the success of the operation, the second stage must be done not later than a month after the first unless, in addition to the slightly higher temperature, there are other indications which necessitate postponement.

The patient is placed on his back with the shoulders raised as high as is necessary for comfort. A vertical incision is made over the centre of the costal

* This is very unusual when the operation is done for bronchiectasis, but may occur sometimes in cases of phthisis.

cartilages extending from immediately below the clavicle to 2 cm. below the costal margin. A second short incision, extending outwards for 3 cm. from the upper end of the first and parallel with the clavicle, will be found greatly to facilitate access to the first costal cartilage. The muscles overlying this part of the chest wall (the pectoralis major and the rectus abdominis) are divided and the underlying costal cartilages fully exposed. The intercostal muscles are detached from the margins of the cartilages, and the pleura and triangularis sterni muscle, together with the internal mammary artery, are separated from the posterior surface. The whole length of the 2nd and 3rd costal cartilages are now removed so as to allow of easier access to the 1st. The removal of this cartilage presents some difficulty and requires considerable care, as the clavicle is firmly bound down to it by the strong broad rhomboid ligament, and close to its posterior surface are the innominate or subclavian artery, the innominate vein and the internal mammary vessels. The rhomboid ligament is divided and the intercostal muscles are cut off from the inferior border. The adjacent structures must then be carefully separated from the posterior surface and the superior border, and the copper spatula, bent like a hook, passed behind the cartilage and brought round over its upper edge. In young adults the cartilages can be divided with a knife, but if ossification changes have taken place, bone forceps will be required. The 5th is the next cartilage excised, the 4th being left as a bridge to prevent too extensive collapse of the chest wall while dealing with the costal margin. The 6th and 7th cartilages are united to a greater or less extent along their adjacent margins, and as there is not room to work between them they are resected together. The muscles attached to the inferior border of the 7th and to the deep surface of this and of the 6th cartilages (transversalis abdominis, diaphragm and triangularis sterni) are separated by knife and raspatory, and a portion of these two cartilages equal in length to that removed from the 5th is excised. The 4th rib alone remains to be dealt with ; this is grasped in a pair of lion forceps and cut off at its junction with the sternum. The chest wall can now collapse and is allowed to do so at a slow rate, this being controlled by the hold on the cartilage. When the collapse is complete, this remaining cartilage is detached from the rib. The gap in the costal margin should now be examined to ensure that the resection has been sufficient to prevent a closer approximation than 1 cm. at the end of inspiration when the cut ends of the cartilages are most nearly in contact.* Unless this gap is left, the further collapse of the lung and chest wall which occurs during the ensuing weeks will cause the outer part of the costal margin to override the inner ; or they will come into apposition and interfere with the completeness of the collapse. The pectoralis major and the rectus muscle,

* When the central parts of the ribs are freed from their attachments in front and behind, they no longer take any active part in the respiratory movements of that side of the chest. During inspiration they and the underlying lung are drawn mesially by the increase in the negative pressure in the opposite half of the thorax, while during expiration the ribs are displaced outwards by the partial expansion of the collapsed lung, air being driven into the bronchi by the expiratory force of the sound lung. This movement decreases and is finally abolished when the dividend ends of the ribs and cartilages become fixed by fibrous tissue and new bone formation.

together with its sheath, are then sutured with catgut and the wound closed. The many-tailed bandage applied over the dressing must not be so tight as to constrict the movements of the sound side of the chest, and yet it must, as far as possible, control the movements of the mobilised ribs. If there is any reaction after this operation, it is advisable not to loosen or change the bandage until the tenth day, as the change in the degree of pressure on the mobilised chest wall may increase the reaction.

After Treatment.

It is most necessary that, in the first few days after the operation, the patient should rid himself, by coughing, of the secretions which accumulate in the bronchi. There is always some part of the chest which is painful even when the alcoholic injection of the intercostal nerves has been systematically carried out ; the aggravation of this pain by violent movements, together with the yielding of the chest wall and the interference of the diaphragm by the detachment of some of its fibres from the costal margin, makes the patient refrain from coughing as much as possible. This difficulty can be overcome by giving morphia and encouraging the patient to cough as soon as the pain subsides. Apart from this, there is no special treatment necessary, but the patient will require careful nursing and gentle handling, with as little movement as possible. Narcotics should be given without hesitation, if the patient has difficulty in sleeping.

It occasionally happens that, as the lung collapses, a collection of pus is prevented from escaping. The symptoms due to this will cause some anxiety until, after a few days, the pus escapes into a bronchus and is expectorated. This happened in one of my cases, but the symptoms disappeared a few days after communication had been established, and did not recur.

For the next two or three months after the operation, the patient should live, as much as possible, in the fresh air, and should avoid all risks of infection. The full expansion of the remaining lung must be encouraged by breathing exercises, as soon as the patient has sufficiently recovered from the immediate effects of the operation.

The resulting visible deformity of the chest wall after this operation is surprisingly small. The position of the clavicle and scapula are not materially interfered with, and the shoulder, therefore, does not drop, nor is the posterior axillary fold affected. The anterior fold is less pronounced owing to the change in position of the ribs and, at first, some wasting of the pectoralis major. The sinking in of the chest wall is more obvious from the front and there is a gap greater than normal between the arm and the body. This deformity is easily hidden by slightly padding the clothes. Some scoliosis is certain to develop, but if the patient is warned and is careful, it is insignificant in degree.

TREATMENT BY LOCAL COLLAPSE OF THE CHEST WALL AND PARALYSIS OF THE DIAPHRAGM.

There are three groups of cases in which the operation of rib mobilisation can be replaced by the somewhat less severe one of local decostalisation. The essential feature common to all three gourps is that the changes are definitely limited to the lower part of the lung. The type of case for which this operation is suitable are those in which :—

1. Bronchiectasis involves the lower lobe only and the dilatation of the tubes is not great.

2. Bronchiectatic changes are limited to the inner part of the lower lobe, *i.e.*, the dilatation is confined to the main descending bronchus.

3. Chronic abscess (single or multiple) is the primary change and the bronchial dilatation is secondary, and the disease is confined to the base of the lung only.

The difficulty in obtaining successful results by this method of treatment is that the wide arch of the ribs immediately above that part of the chest which is decostalised, and the comparative rigidity of them, interferes very greatly with the falling in of the chest wall. In order, therefore, to obtain a reasonable degree of collapse of, for instance, that part of the lung under cover of the 7th, 8th and 9th ribs, it is essential that the 5th and 6th ribs should be removed in addition to the three or four lower ones. It is clear, therefore, that when the disease extends as high as the 5th rib in the mid-axillary line, the rib resection becomes so extensive and complicated by the difficulty of securing access to those parts covered by the scapula that the seriousness of the operation approximates closely to that of rib mobilisation, without giving the advantages of the extensive collapse which that procedure ensures.

In every case the phrenic nerve should be divided in the neck, so as to paralyse the diaphragm on the affected side and secure thereby a still greater degree of diminution of the base of the pleural cavity.

The *technique* of the operation of decostalisation has already been sufficiently described on pp. 70 and 78, and that of section of the phrenic nerve on p. 192. The whole of each rib extending from the tubercle behind to the costo-chondral junction in front should be taken away. After the wound is healed the patient must be fitted with a special pad maintained in position by straps over the opposite shoulder and round the trunk, so as to keep the chest wall firmly collapsed until the regeneration of the bones is complete.

TREATMENT BY LIGATURE OF A BRANCH OF THE PULMONARY ARTERY.

This method of treatment is recommended by Sauerbruch as the result of experimental work done by him and Bruns. They found that "a peculiar process of induration develops after tying a branch of the pulmonary artery, as a result of which the organic structures of the lung are very considerably changed. There is an extensive connective tissue proliferation, especially from the interstitial tissue. The individual alveoli are surrounded and compressed. Later the granulation tissue begins to contract. At the same time strong adhesions form between the visceral and parietal pleura, and these are strictly limited to that area supplied by the ligatured artery."* The same change occurs in man and the shrinkage of the fibrous tissue is so great that it necessitates the mobilisation of the overlying chest wall.

My own experience tends to show, however, that the fibrosis of the lung is not permanent, and that after a time (eighteen to twenty-four months) the alveoli open up again and functionate. This is illustrated by the case of a patient, aged 17, who had brochiectasis for seven years with attacks of retention of sputum associated with fever and great offensiveness of the breath, general wasting and progressive weakness. The bronchiectasis was confined to the lower lobe, and I ligatured the branch of the pulmonary artery supplying this lobe in June, 1913, removing at the same time part of the overlying ribs. The lower lobe fibrosed and contracted and the patient improved so much that in March, 1915, he joined the Army and for six months was able to do all the physical training and other work required. This strenuous life produced, however, some slight return of the cough and sputum, and he was not passed as fit for service abroad. The sputum is small in quantity, is no longer offensive, and in no way interferes with his normal work, nor does it appear to affect his general health. The radiogram taken January, 1917 (Fig. 76), shows that there is a considerable return of free air-way into his lower lobe, and a striking diminution in the extent of the former shadows caused by the dilated bronchi and in the opacity due to the fibrosis of the lower lobe which developed after the operation.

It is extremely difficult to estimate the value of this operation. The number of cases operated on by Sauerbruch is small and the patient referred to above is the only one who, to my knowledge, has been so treated in this country. The early results of this method of treatment have been good, but Sauerbruch has not given any account of the progress of his cases during the three to five years subsequent to the operation, so that it is not possible to determine how permanent is the benefit obtained by ligation of the artery.

This operation is suitable for cases of bronchiectasis strictly limited to the lower lobe. As the ligature of the artery is followed by extensive fibrosis,

* "Tecknik der Thoraxchirurgie." Sauerbruch and Schumacher. Berlin, 1911.

Fig. 75. Bronchiectasis of the right lower lobe. The artery to that lobe had been ligatured, June, 1913. Radiogram taken six months later. Note the opacity due to the fibrosis in the lower lobe.

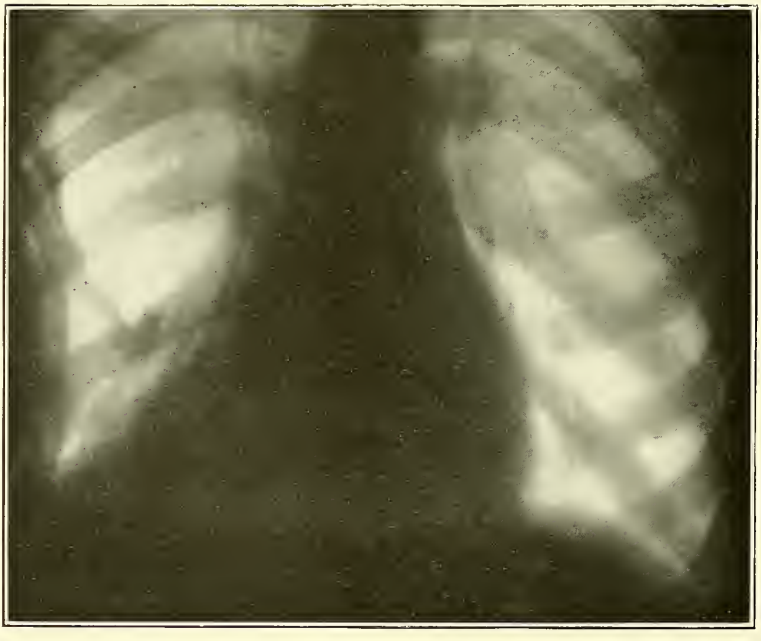

Fig. 76. Same case, January, 1917. The opacity of the lower lobe has disappeared. This patient did his full course of training in the Army from March to August inclusive, 1915.

it is necessary to allow for the shrinkage in volume of this part of the lung by removing parts of the underlying ribs and possibly by paralysing the diaphragm also. The operation, moreover, is not free from difficulties and complications, some of which may be serious. If the pleural membranes are closely adherent, separation of them, and consequently access to the artery, may be impossible. A pleural effusion is a frequent complication whenever there has been any considerable intrapleural manipulation. Such effusion may not exercise any harmful influence in the recovery of the patient, but should there be any sloughing of the lobe, as happened to the case referred to above,* an empyema will result and the consequence may be extremely serious.

The evidence available up to now does not show that the operation of ligature of a branch of the pulmonary artery possesses any advantages over that of rib mobilisation or over that of partial decostalisation combined with section of the phrenic nerve.

Technique of the Operation.

The patient is anæsthetised by the insufflation method, and is placed on the table lying on the sound side, with the arm of the affected side drawn upwards and backwards. Sauerbruch describes the operation as follows :— "The chest wall is opened in the typical way by an incision through the 5th intercostal space.† Since in many cases adhesions exist between the surface of the lung and the chest wall, the separation of the pleural membranes must be done with the greatest care so as to avoid injury to the lung. When there are no adhesions, orientation is easy. The lower and upper lobes are carefully separated and the pedicle of the lower lobe is exposed. Three structures will be found here running into the lung in close proximity with each other : the artery, the bronchus, and the vein. They are easily differentiated when one knows their anatomical relationship. The bronchus lies in the middle between the two vessels, the vein below and the artery above. The artery is the first structure seen on exposing the pedicle. No other method of orientation is advisable. The artery should not be sought for by its pulsation, as transmitted is easily mistaken for true pulsation . . . The isolation of the artery is somewhat more difficult when the pedicle is short and the upper lobe has to be separated off it. The free exposure of the pedicle is effected with the least damage to the lung tissue by blunt dissection with gauze. It must always be remembered that in many cases the branch of the artery to the left lower lobe bifurcates high up and it is then necessary to isolate and tie the two individual branches." A ligature is passed round the vessel with an aneurism needle and tied tight. The lung is expanded by raising the intratracheal pressure, and the wound is closed (see p. 59).

* Sauerbruch states that sloughing of the lobe has not followed ligature of the artery, either in his patients or in the animals on which he has experimented.

† See p. 237.

It is essential that the further shrinkage of the lung due to the contraction of the new fibrous tissue be facilitated. Already, prior to the operation, the surrounding walls have become as closely approximated as possible. A greater diminution of the lower part of the pleural cavity can be obtained by surgical means only. It will be necessary, therefore, about a fortnight after the operation, to remove portions of the 5th to the 10th ribs. The reduction in size of the pleural cavity can with advantage be supplemented by paralysing the diaphragm by dividing the phrenic nerve.*

The possibility of a pleural effusion, of sloughing of part of the lobe and of empyema must be borne in mind.†

Prophylaxis.

It has been pointed out in the beginning of the chapter that bronchiectasis is not a disease *sui generis*, but a sequela generally of some acute disease, which, having failed to clear up completely, persists in the form of an insidious chronic inflammatory process. The primary result of this is a progressive fibrous tissue formation, which in turn, sooner or later, leads to bronchial dilatation. If the lung was not enclosed by rigid walls, but was free to collapse with the contraction of the fibrous tissue, there would be no dilatation of the bronchi. If, therefore, when a fibrous change is definitely in progress and before this has had time to affect the walls or lumen of the bronchial tubes the extent to which the lung can contract is increased, there is a probability that the inflammatory processes responsible for the fibrosis will have subsided before the new limit of free contraction of the lung is reached ; and a certainty in such case that the adverse influence of the fibrous tissue on the walls of the bronchi will be prevented.

It is for this reason that it is strongly urged that in all cases of pneumonic consolidation in which there has been delayed resolution and in which the X-rays show the presence of fibrosis, specially of the lower lobe, section of the phrenic nerve in the neck should be done. This will produce paralysis of the diaphragm on the same side ; the muscle will rise from 2·5 cm. to 4 cm. into the thorax, and the lower part of the pleural cavity will be considerably diminished in size. If, moreover, the shrinkage of the lung is greater than has been allowed for by this change, the pull of the contracting fibrous tissue will now be exerted on a flaccid muscle which will yield, instead of on rigid structures.‡ The change in the position of the diaphragm produced by section of the phrenic nerve in the neck is shown in Figs. 63 and 64.

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* See p. 192.

† The treatment of bronchiectasis by pneumotomy and drainage, by pneumectomy or by extrapleural replacement by fat, is not discussed in this book, as the author does not consider these methods to be satisfactory in comparison with those detailed above.

‡ The technique of this operation is described on p. 192.

Cerebral Abscess and Intrathoracic Suppuration.

In the chapters dealing with empyema and with gangrene and abscess of the lung, cerebral abscess was referred to as a complication of these diseases. It occurs also, and relatively more frequently, as a complication of bronchiectasis. This combination of lesions was very fully investigated by Schornstein, whose thesis was published as the first Shornstein lecture ;* from this the following facts are taken : Of 69 collected cases of cerebral abscess associated with disease of the lung or pleura, the intrathoracic lesion in 55 per cent. of the cases was bronchiectasis ; in 22 per cent. was empyema ; in 11·5 per cent. was gangrene and abscess, and in 4·5 per cent. was tubercle. Moreover, in bronchiectasis, abscess of the brain is, next to broncho-pneumonia, the most frequent cause of death. It was the terminal factor in about 20 per cent. of 63 collected post-mortems on patients suffering from bronchiectasis. " Bronchiectasis is the most frequent antecedent of cerebral abscess, and cerebral abscess is the second most common cause of death in bronchiectasis."

" Cerebral abscess has occurred with empyema both in cases which have been operated on and in cases which have been untouched. Alike where the pus in pleura was undiscovered or simply tapped or resection of rib performed, cerebral abscess has followed . . . . In the great majority of instances, cerebral abscess develops long after, sometimes many weeks after, the resection. It generally occurs in cases where the opening has never properly closed, and a more or less continuous or intermittent purulent discharge has been kept up, but occasionally where the resection opening has already properly closed."

Cases of cerebral abscess have also been recorded as developing after irrigation of the pleural cavity. Considering the frequent association of cerebral abscess and bronchiectasis, it is surprising how rarely such a lesion of the brain complicates pulmonary tuberculosis.

The abscess may be multiple, but is more often simple, and is usually in the left hemisphere (in about 76 per cent. of the cases). The frontal or the parietal lobes, and next to them the occipital lobes, are the most often affected.

Schornstein considers " that these abscesses are produced by the action of the tiny thrombi dislodged from small branches of pulmonary veins, carried to the left auricle and left ventricle direct, and so into the aorta."

The symptoms are more often of the acute than of the chronic type ; " from the onset of the first cerebral symptoms till death is usually a very short period." In the cases recorded " the duration of cerebral signs till

* Thesis for the degree of M.D. Oxon. Schornstein lecture published in *The Lancet*, December 18th, 1909.

death varies from three to twenty-eight days, and the average is ten days." Whilst the symptoms are usually clearly indicative of the nature of the lesion, the localisation of it is, as a rule, much more difficult owing to the comparative frequency with which the silent areas are involved.

"Operation for the evacuation of abscess in cases where localisation is possible is clearly indicated, but the chances of effectively relieving the patient are very small. The general ill-health precludes any real possibility of success."

Chapter X.

Streptotrichosis of the Lungs and Pleura.

Of the granulomata affecting the lung, streptotrichosis and syphilis are in striking contrast to tuberculosis in that they are of comparatively rare occurrence ; but the similarity of the clinical picture of all three infections is at times so great that the differential diagnosis from tuberculosis can be made only by the detection of the ray fungus in the sputum in the one case, and by a positive Wassermann reaction and a rapid and sustained improvement under iodide treatment in the other. The smallness of the number of recorded cases of infection by the streptotricheæ gives an erroneous conception of the frequency of the disease. There are undoubtedly a considerable number of cases reports of which have never been published, and possibly a still larger number in which the correct diagnosis has never been made.

Several varieties of streptotricheæ have been described ; in some (actinomycoses) the ray fungus is so well developed as to be easily recognised ; but in others the threads are slender, show but little tendency to branching and break up rapidly into segments resembling Koch's tubercle bacillus. Foulerton maintains that there is a close relationship "between certain undoubted species of streptotricheæ and the parasite or parasites of tuberculosis. Our own investigations into the pathology of streptothrix infections, together with much work bearing on the subject which has been carried out elsewhere, have led to a very definite conclusion as to the relationship, amounting to actual generic affinity, between this class of infections and those recognised under the name of tuberculosis."*

The streptothrix infection may be saprophytic or pathogenic ; the former is often found in pulmonary lesions in which there is cavity formation. The pathogenic produces a disease *sui generis* and it is this variety only with which we are concerned.

The disease appears as a primary infection of the lung or of the pleural membranes, or as a secondary infection by direct extension from a neighbouring focus, most frequently in the liver. The lower lobe is the part of the lung commonly affected ; infection of the upper lobe is rare.

The morbid anatomy changes in the lungs are closely similar to those found in cases of necrosing broncho-pneumonic tuberculosis or of chronic

* "The Streptotrichoses and Tuberculosis."

interstitial pneumonia, or of miliary tuberculosis. When the pleural cavity is involved, there may be great thickening and fusion of the membranes, or the two layers of the pleura may be separated by a purulent effusion, or by a firm spongy vascular mass in which there are multiple abscess cavities. The pus from the pleural cavity is yellow or orange in colour, and contains, when the fungus is of a well-developed variety, bright yellow nodules; it has frequently an offensive odour. The sputum, when abundant, may show the same characteristics.

Clinically, the streptothrix infections are divisible into two main groups: (*a*) in which the symptoms and signs are mainly pulmonary; (*b*) in which they are mainly pleural.

The Pulmonary Group.

The clinical varieties conform with the morbid anatomy changes described above. There is the type which resembles caseating tubercle; that which resembles miliary tubercle; and a third which suggests a localised chronic interstitial pneumonia. When the lesion is in the upper lobe, diagnosis is rendered still more difficult. If tubercle bacilli cannot be found in the sputum of a patient suffering from a disease the symptoms and signs of which suggest active pulmonary tuberculosis, an examination should be made for acid-fast organisms: the presence of tubercle bacilli does not exclude a streptothrix infection. Repeated attacks of pain due to associated pleurisy are a characteristic, but not necessarily constant, feature of this disease. Until the necrosing areas communicate with a bronchus, the cough is infrequent and the sputum scanty: the symptoms in the earlier stages are mainly constitutional.

The Pleural Group.

There are two striking features of pleural streptotrichosis: they are pain and the presence, sooner or later, of one or more subcutaneous abscesses. These, if left, ulcerate through the skin and are replaced by chronic sinuses. The abscesses develop most frequently in the upper part of the chest. As a rule, no track can be found leading from the subcutaneous collection of pus through the chest wall into the empyema. Sometimes there is necrosis of the rib underlying the abscess. Œdema of the chest wall is common even when there is no extrapleural pus.

The pulmonary type of case may run its course without producing any changes in the pleural cavity other than great thickening and fusion of the membranes; whilst the pleural type, though invading the chest wall and causing necrosis of the ribs and discharging sinuses, may produce no pulmonary symptoms other than slight cough and a little mucoid expectoration. There are some cases, however, in which the streptothrix infection is much more virulent. It may begin in the lung and produce necrotic changes; it then passes on to the pleura, causing fibrosis and localised abscess formation, and

later invades the chest wall, destroying bone, cartilage or soft tissues, and spreading in all directions along the planes of least resistance.

The disease runs a rapid course. The very acute forms destroy the patient in from six to nine months from the onset of definite symptoms; the more chronic varieties, unless checked by treatment, take from twelve to eighteen months. It is probable, however, that the infection is present for some weeks before any localising symptoms or signs appear; during this period, the patient suffers from malaise, but is not definitely ill.

The following accounts of two cases are fairly characteristic of the pulmonary and of the pleural type of the disease.

The first patient was a man aged 48. From October, 1909, to July, 1910, he had a constant slight irritating cough and repeated attacks of pain in the cardiac region associated with shortness of breath. He felt ill, but was able to continue his work most of the time. From July to December he was under the care of Dr. Kincaid. There was shortness of breath, cough with but little expectoration, some epigastric pain and an evening rise of temperature up to 100° or 101° F. In September, a pint of clear fluid containing flakes of lymph was removed from the left pleural cavity. No tubercle bacilli could be found in the sputum or in the fluid. In November, when I first saw the patient, he was not so well, the cough was persistent and the sputum scanty. The maximum impulse of the heart was in the left nipple line. The lower part of the left side of the chest was contracted and there were signs of fibrosis in the lower lobe. There was no clinical or radiological evidence of fluid. The upper lobe and the right lung were healthy. The sputum was found to contain numerous fine branching threads and a few clubs. From reports received subsequently, the disease made steady progress but did not directly invade the pleural cavity. The patient died in the spring of 1911.

The second patient was a man aged 25.* In June, 1916, a swelling appeared in the right loin. The right kidney was explored, was found to be infiltrated with pus and was drained. He then developed a cough and a muco-purulent sputum; there was pain in the left side of the chest and in the left shoulder. When admitted to University College Hospital on November 29th, the patient was seriously ill. He was cachectic and his temperature showed a daily variation of from 3 to 4 degrees. There were numerous scars and sinuses over the right loin and over the lower part of the chest on the same side. The tissues over the lower part of the left chest were œdematous. The right lung was normal. The left lung was partially compressed and numerous rales were audible over it. There was a localised empyema in the left axillary region (see Fig. 77). The pus from the pleural cavity was yellow in colour and contained numerous granules due to the ray fungus; the streptothrix was obtained also from the sinuses in the right loin. An

* This patient was seen by me in consultation with Captain Wright and Mr. Shattock.

attempt to evacuate the empyema was unsuccessful as the patient collapsed whilst the local anæsthetisation of the chest wall was being done ; he died a few days later.

Fig. 77. Streptotrichial empyema.

At the post-mortem it was found that there was no pus in the lung. At the back of the right pleura there was a thick fibrous mass containing opaque orange-coloured pus. This mass extended down along the spine and was continuous with another larger mass which occupied the space between the right and posterior surfaces of the liver and the diaphragm and continued into the right flank almost to the iliac crest. There were universal fibrous peritoneal adhesions ; the cæcum was normal.* The appendix had been removed eighteen months previously, but no information as to the condition of this organ could be obtained.

TREATMENT.

The experiences of recent years have shown that the outlook of these cases, especially if the disease is recognised in its earlier stages, is not hopeless. Several cases have been cured by the use of iodides and of autogenous vaccines.

* Extract from the notes of the post-mortem made by Mr. Lawrence.

Potassium iodide should be given as soon as the diagnosis has been made. The dose at first is 20 grains three times a day, but this is increased rapidly until the patient is taking 60 grains three or four times a day. This drug must be continued for some weeks after the symptoms have completely subsided, the dose being diminished gradually before it is finally omitted. A few cases of streptothrix infection have been treated with completely satisfactory results by the use of vaccines only. It is advisable, however, to combine the injections with the iodides.

It is doubtful whether any known treatment is of value in the fulminating types of the disease. The drastic operative measures which have been recommended by some surgeons have never proved successful. Operative intervention should be limited to the pleural cases only—to the treatment of the intrapleural disease and of the subcutaneous abscesses.

When the localised disease involves the pleural membranes there may be a collection of pus shut off from the rest of the pleural cavity by a dense wall of fibrous tissue ; or the pleural cavity may contain a spongy fibrous mass of breaking down granulation tissue in which are enclosed a number of small pockets of pus.

It is not, as a rule, possible to determine clinically which of these two conditions is present. Occasionally a correct diagnosis can be made with the aid of a good radiogram. If the plate shows a circumscribed shadow of variable density between the lung and the chest wall, the presumption that the pus is contained in a series of loculi is probably correct. A uniform shadow does not, however, necessarily mean that the abscess is unilocular. It is of great importance to recognise which type of condition is present, as the treatment of each differs considerably, but it must be clearly realised that the recognition of the true state of affairs may not be possible until the attempt to evacuate the pus by aspiration has been made.

Unless there is evidence that the abscess is multilocular, treatment by aspiration with oxygen replacement must be tried. In this way the whole of the pus can be evacuated from a single cavity. Fluid will, however, re-accumulate, and it is necessary to repeat the aspiration at first every few days, but later at longer intervals. At the same time, iodides in increasing doses must be given, and it is advisable also to give injections of an autogenous vaccine.

Aspiration, when the disease consists of multilocular abscesses, is of very little use. One only, or perhaps two, of the cavities will be evacuated, while the others, together with the degenerating granulomata, remain untreated. In this type of case it is necessary that the pleural cavity should be opened in order that direct access to the diseased area may be obtained. This exposes the patient to the very considerable risk that the wound in the parietes may become infected, and to the added dangers and distress which are the inevitable result of a chronic sinus and of secondary infection. Certain

precautions must therefore be taken before, during and after the operation of pleurotomy so as to prevent as far as possible the occurrence of these complications.

For two or three weeks before the operation, unless the case is urgent, the patient should be given large doses of potassium iodide and a series of injections of an autogenous vaccine. During the operation the wound must be continuously protected by gauze soaked in an antiseptic solution, and at the end of it the deep and superficial margins of the whole wound must be brought into the closest apposition by sutures.* After the operation, the fluid that re-accumulates must be repeatedly aspirated — as high an intrapleural negative pressure as can be tolerated with comfort being left on each occasion. This aspiration is necessary in order to prevent the fluid coming in contact with the wound and being forced into the cellular tissues of the chest wall when the patient strains or coughs. The rate of accumulation of the fluid is always most rapid during the first few days after the operation. This is particularly the case during the ensuing twenty-four to forty-eight hours, and it is very probable that the first aspiration will be necessary on the day after the operation.

When the pleural cavity has been opened, the free access to the diseased area admits of more extensive local treatment than the simple removal of the pus. The barriers between the various loculi should not only be broken down but should be removed, together with all the debris of degenerated tissue. The surrounding walls must be scraped with a sharp spoon and the cavity that is left thoroughly cleaned out with mops. There may be a considerable amount of bleeding, but this is readily checked by packing the cavity with gauze. The gauze is not removed until the greater part of the wound has been closed by sutures.†

It is most important that in those cases in which an empyema is complicated by a subcutaneous abscess, the intrapleural collection should not be evacuated through the superficial abscess, as this would certainly lead to the secondary infection of the pleural cavity. The subcutaneous abscesses are opened and scraped and necrosed portions of rib are removed. Not only is no attempt made to find the track leading into the pleural cavity, but special care must be taken to avoid injuring the pleural membrane. These abscess cavities are packed with gauze soaked in some strong antiseptic and free drainage of them is maintained until the spaces are filled with healthy granulation tissue. The empyema is treated either by aspiration with oxygen replacement or by pleurotomy through healthy tissues, but in neither case is a negative pressure higher than equal to 2 mm. of mercury produced, lest infected material from the superficial abscesses be sucked into the pleural cavity.

* Catgut is, of course, used for all but the cutaneous sutures.

† The methods of making the opening into the pleural cavity, and of closing the wound, are described on p. 59.

Chapter XI.

HYDATID DISEASE OF THE LUNGS AND PLEURAL MEMBRANE.

The tænia ecchinococcus has two stages in its life history—the larval stage which is spent in sheep and occasionally in man, and the adult or worm stage which is passed in the intestinal track of the dog. The ova of the parent worm escape with the excreta of the dog and are conveyed to the stomach of the intermediate host in the water or the food. The chitinous envelope is digested by the gastric juice and the embryo is set free. The embryo, by means of its hooklets, penetrates through the wall of the stomach and travels directly, or by way of a vein, usually into the liver, and occasionally into the lung. There the parasite lodges, loses its hooklets and develops into a cyst (the hydatid cyst), which is lined with an external translucent laminated ectocyst and an internal granular endocyst.

The hydatid contains a non-albuminous, slightly saline clear fluid. By changes in the endocyst, daughter cysts and brood capsules are formed ; from these latter develop scolices which are the immature heads of the adult worm. The cysts are occasionally sterile.

More than half the number of hydatids are found in the liver. The second commonest organ in which they occur is the lung, but the number found here represents about 10 per cent. only of the total. Hydatids of the pleural membranes are extremely rare. It is obvious, from the life history of the parasite, that hydatid disease occurs most frequently among men whose work brings them into close contact with dogs and sheep.

The irritation set up by hydatids which develop in the liver and most other solid organs causes a reaction of the tissues and the formation of a fibrous wall around the cyst. This change is not found in the lung, where the only evidence of irritation is a certain increased vascularity of the part.

The symptoms and signs depend on the rapidity in the growth of the cyst and on the increase in pressure which occurs within its walls. This renders it particularly liable to rupture into a bronchus, when a spontaneous cure may result ; more frequently, however, the cyst becomes secondarily infected.

The Unruptured Hydatid.

When the cyst is small, is deeply situated, and particularly if it is sterile and of slow growth, it may give rise to no symptoms. Evidence of some pathological change in the lung may then be discovered as a result of routine

examination of the chest, or by radiography only. A rapidly growing cyst produces, on the other hand, a chronic cough, slight mucoid expectoration occasionally tinged by streaks of blood, and dyspnœa. If the hydatid is near the surface of the lung, there is irritation of the pleura and pain. The physical signs indicate the presence of a solid body in the substance of or at the surface of the lung. The radiographic appearance of an uncomplicated cyst is well shown in Fig. 78.

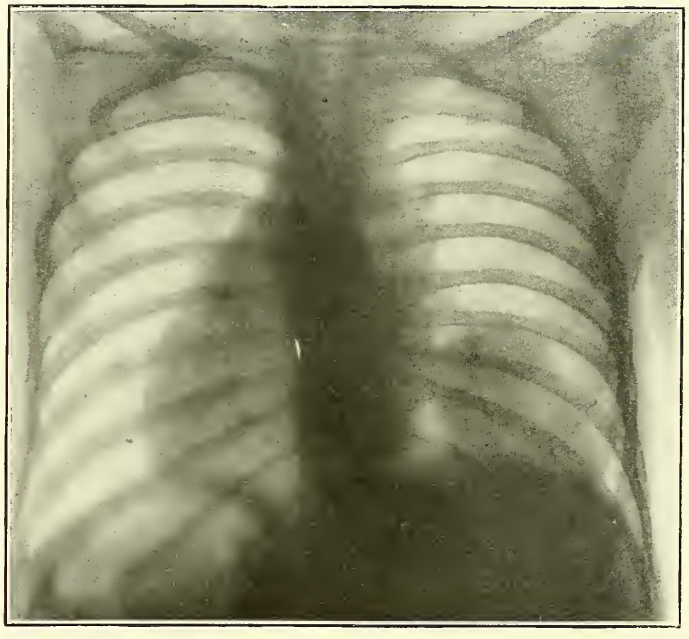

Fig. 78. Hydatid of the lung. *Radiogram by Dr. Ironside Bruce.*

Fever or any considerable disturbance of the patient's general health is rare. Eosinophilia is frequently but not constantly present. A positive serum reaction is diagnostic, but a negative result is inconclusive evidence. Antibodies are said to be found in 95 per cent. of cases.

The Rupture of the Cyst.

The cyst may rupture into the pleural cavity, into the pericardium, into the peritoneal cavity or into a vein; in at least half the cases, the rupture occurs into a bronchus. There is then a great and immediate danger of death by suffocation. If the patient escapes death, he will cough up the contents of the cyst, and occasionally the lining membrane also. In such a case a spontaneous cure may result. During this period there is, however, the danger of impaction of the cyst wall in the larynx, or of a severe hæmorrhage from the lung during the process of separation of the membrane.

The Ruptured Hydatid.

When the lining membrane does not spontaneously separate and escape, infection of the cavity occurs, and the symptoms and signs of a chronic abscess develop. The appearance in the radiogram depends on the efficiency of the drainage. The cyst may be completely filled with pus and give a dense rounded shadow with a surrounding area of pneumonic consolidation. When the cavity is empty, there will be an opaque ring corresponding with the walls of the cavity and surrounding an abnormally translucent area. A cyst which is partially filled with pus will show an opaque shadow in the lower part, whilst the upper portion will be translucent. The line between these two is sharply defined and horizontal.

TREATMENT.

The Unruptured Cyst.

A small hydatid, which is latent as regards symptoms, does not require immediate treatment. It must be kept under constant observation, and radiograms should be taken periodically to note if there is any increase in size. As soon as there is definite evidence that the cyst is enlarging, it should be treated without further delay.

Treatment of a hydatid by aspiration must never be attempted owing to the intracystic pressure and the tendency it has to rupture. Exploratory puncture is equally dangerous unless the patient is anæsthetised by intratracheal insufflation and the operation has proceeded to that stage at which it is possible immediately to incise the cavity so as to allow the contents to escape externally.

Exposure of the cyst, incision into it and drainage is the only certain and by far the safest means of treatment, and this method should be adopted in all cases of hydatid cysts which are producing symptoms, or which, though symptomless, are increasing in size. The patient must be anæsthetised by the intratracheal insufflation method, the air and anæsthetic being administered at a fairly high pressure. This helps to diminish the danger of intrabronchial rupture by reducing the difference in pressure on the inner and outer walls of the cyst. The route of access is through that part of the chest wall which is nearest the tumour. If the cyst is small, it will suffice to make an incision through the skin and muscles and to remove a portion of the overlying rib. When the hydatid is large, it is advisable to turn back a flap of the soft tissues covering three or more ribs and to remove the exposed portions of bone. The greatest care must be taken during this and the subsequent manipulations that the least possible pressure is exerted on the underlying soft tissues. The two pleural membranes should be brought into close apposition by a line of sutures encircling the exposed area (the method of doing this is described and illustrated on p. 132). It is at this stage that it is permissible, if there is any doubt as to the location of the hydatid,

to use an exploratory puncture. If, however, the cyst is superficial and it is known to be immediately beneath the wound, it is safer to incise the posterior layer of the periosteum, the pleural membranes, the superficial lung tissue and the wall of the cyst with a sharp knife. The rush of the escaping contents of the hydatid should be controlled by the finger, lest the rapid alteration in pressure leads to an acute congestion of the surrounding lung parenchyma, and so causes hæmorrhage. The endocyst is, as a rule, easily detached and should be removed, but no attempt must be made to strip off the ectocyst from the surrounding lung parenchyma, as this would cause severe bleeding. The margins of the incision into the ectocyst are stitched up to the wound in the pleura to ensure free drainage of the cavity. A large drainage tube is inserted, and the flap of skin and superficial tissues is replaced. The end of the tube may be brought out through a fresh incision in the centre of the flap or through the original incision. Gauze soaked in some antiseptic is placed round the tube where it is in contact with the skin to prevent secondary infection. The cavity in the lung heals by granulation and by approximation of the walls. At the end of five days the tube is shortened ; it can usually be dispensed with at the end of ten days.

The Ruptured Hydatid.

It has already been stated that if rupture into a bronchus is followed by expectoration of the endocyst, a spontaneous cure may result ; surgical intervention is required usually for those cases only in which secondary infection has occurred ; that is, for those cases in which the hydatid cavity has become a chronic abscess. The treatment of an infected hydatid is exactly the same as is that for chronic interstitial abscess and has been fully described on pp. 136 to 140. The external drainage of an infected cyst which communicates with a bronchus may result in the formation of a chronic pleuro-bronchial fistula, for the closure of which a second operation will be necessary (see p. 81).

Urticaria, due to the absorption of toxins from the contents of the cyst, is a symptom which may appear five to seven days after the cyst has ruptured, or after it has been opened and drained. It does not affect the prognosis and requires symptomatic treatment only.

Hydatid of the Pleura.

The operation for the exposure of a hydatid of the pleura is the same as that for the intrapulmonary cyst. The hydatid which grows in the intrapleural space separates the two membranes from each other. It is equally important in this case as it is in that of the intra-pulmonary cyst, to prevent escape of the contents into the free pleural cavity, if such exists. The walls of the hydatid are, therefore, stitched to the parietal pleura in the same way as is recommended when firm union of the two layers of the pleura is required. The cyst is then incised and drained.

Chapter XII.

Primary Tumours of the Lung and Mediastinal Dermoids.

Tumours of the Lung.

There are three groups of malignant tumours of the lung : (1) Those which are secondary to carcinoma or sarcoma in some other part of the body ; (2) those which start in the hilum or in the mediastinum and spread thence into the lung ; (3) those which originate from the epithelium of the smaller bronchi and involve the hilum and the pleura at a comparatively late period in their development. They are rare, but they are the only ones which are accessible to surgical treatment, and it is this group, therefore, which is dealt with here.

Macroscopically, these carcinomata are always sharply defined from the surrounding lung tissue (see frontispiece). In the nodular variety there is at first a single tumour ; secondary nodules appear later, separated from each other by lung tissue. As the tumours increase they coalesce, the intervening lung tissue disappears, and necrotic changes take place in the centre of the mass. The infiltrating variety forms from the first a homogeneous mass, but degeneration changes may occur in the centre of this also. When the central necrotic area opens into a bronchus, the degenerated tissues escape and a cavity forms which becomes infected from the bronchus.

Microscopically, the tumours are usually squamous-celled carcinomata which originate from the epithelium lining the bronchi ; more rarely, they are cylindrical and start then in the epithelium lining the bronchial glands.

Carcinomata of the lung are symptomless until they reach the surface and irritate the pleura, or until they press upon or establish communication with a bronchus. This latency enables them to grow to a considerable size before there is an opportunity of making a diagnosis. Shortness of breath is probably the earliest symptom, but wasting is occasionally the first indication that there is something seriously the matter with the patient. Cough and mucoid expectoration may appear before there is any direct communication between the growth and a bronchus. As soon as this occurs, however, the cough increases and the sputum becomes muco-purulent and may contain streaks of blood and fragments of tumour. At times the sputum resembles prune juice or strawberry juice in its colour ; this is due to alteration in the blood in the cavity of the growth, and is suggestive, but not

pathognomonic, of a malignant tumour. In the expectorated matter spherical cells which are of considerable size and contain fat granules, together with irregular-shaped epithelial cells with tail or club-like processes, can, as a rule, be found, but repeated examination may be necessary ; these are pathognomonic of carcinoma of the lung. Pyrexia is absent unless there is disintegration of the central part of the tumour or secondary infection.

When the tumour reaches the surface, it irritates the pleural membranes and causes either a painful dry pleurisy or an effusion which may give rise to pressure symptoms. The effusion is at first simple in character, but later is frequently blood-stained.

Physical signs do not appear until the tumour approaches the surface, interferes with the air entry along one or more of the bronchi or discharges

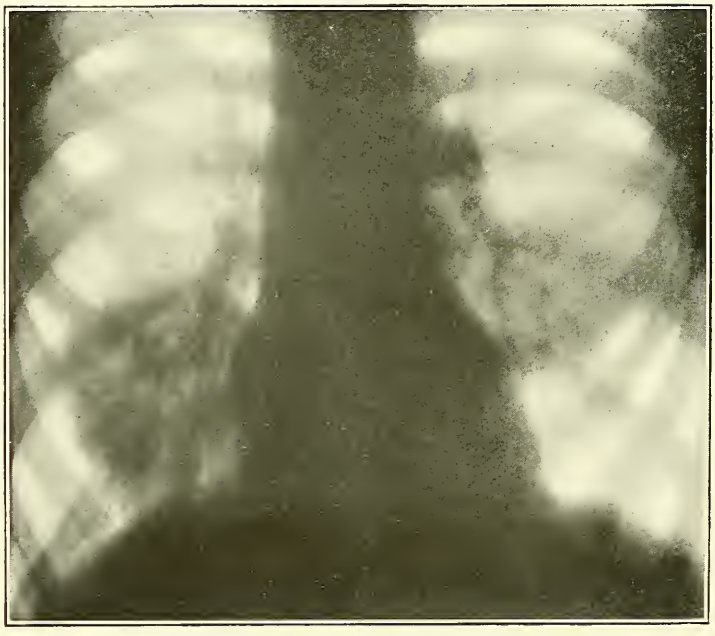

Fig. 79. Primary bronchial carcinoma of the lung.

the products of disintegration into a bronchus. Even when present they may be masked by the signs of some concurrent disease such as bronchitis or emphysema.

The radiographic appearance is shown in Figs. 13 and 79. The outline of the tumour is fairly well defined and there is no surrounding pneumonic consolidation such as is seen round shadows due to an inflammatory lesion. The outline is not, however, so sharp nor is the general shadow so uniform in density as in cases of unruptured hydatid disease.

In the author's case (Fig. 79 and frontispiece), the diagnosis was made entirely on the radiogram. The history of this case is as follows :—F. R., male, aged 44, had had attacks of bronchitis associated with pain and a feeling of tightness in his chest for eight years. These symptoms had become worse during the last six weeks. The patient was examined with the X-rays as part of the routine investigation, and the tumour shown in Fig. 79 was found. No physical signs other than those which could be attributed to bronchitis were detected until three weeks later, although the patient was repeatedly examined by several physicians. Prune-juice sputum was expectorated for the first time a week after the radiogram was taken, and in this sputum numerous large spherical cells with club and tail-like processes were found. The patient refused operation until two months later, when his general and local condition (including the bronchitis) were very much worse. Amputation of the lower lobe was done (see frontispiece). For the first six days after the operation, the patient made good progress, but he then developed an empyema and died. At the autopsy, no evidence of glandular or other secondary infection could be found. The tumour was a squamous-celled carcinoma.

TREATMENT.

Success in the treatment of bronchial carcinoma depends mainly on the early diagnosis and also on the strict observance of the essential points of the technique of the operation.

These points are : the administration of the anæsthetic by intratracheal insufflation (or the use of some efficient apparatus for the compensation of the pneumothorax) ; the adequate exposure of the root of the lung ; the infiltration of the vagus nerve immediately above the hilum with 2 per cent. novocaine so as to reduce shock ;* the treatment of the stump of the bronchus so as to avoid infection of the pleural cavity, and the drainage of the cavity for two days after the operation.

The patient is given morphia, is anæsthetised with chloroform and the intrapulmonary pressure is raised by intratracheal insufflation. A long incision is made through the 5th intercostal space from the outer edge of the latissimus dorsi muscle behind to the internal mammary artery in front. The 4th and 5th ribs are widely separated by strong mechanical retractors. If additional space is required, the 4th and 5th cartilages can be divided. Any adhesions which there may be between the two surfaces of the pleura and between the affected and the adjacent lobe are divided. It is at this stage that the vagus nerve should be injected with novocaine (its situation is described on p. 18 and shown in Fig. 8). The vessels and the bronchus supplying the affected lobe are exposed by blunt dissection, and the former

* As a result of experimental investigation on animals, the author has found that the intrathoracic anæsthetisation of the vagus completely abolishes the shock of manipulations on the lung. In the patient referred to above, the vagus was injected with novocaine and the post-operative shock was almost nil.

are divided between double ligatures. The lung tissue at the base of the lobe is cut through and any bleeding vessels are picked up and ligatured. The lobe is now attached to the hilum by the bronchus only ; a purse-string suture is passed round the bronchus through the outer coats, and the tube is crushed by powerful forceps applied immediately distal to the suture. The crushed section is divided with a cautery and the lobe is removed. The proximal cut end of the bronchus is then invaginated and the purse-string suture tied. The adjacent divided surface of the lung is drawn over the stump and fixed in place with a few sutures as a further protection for the pleural cavity from infection. A small drainage tube is passed through a separate puncture incision in the lower part of the cavity, so as to remove the effusion which will occur during the ensuing day or two. The wound is then closed in the manner already described on p. 59.

While the drainage tube is *in situ*, it must be well covered with dressings so as to prevent inspiration of air ; for the same reason it must be clamped if a change of dressing is necessary. The tube should be taken out on the second day and the puncture wound will become obliterated during the next twenty-four hours. Any further effusion which may take place into the cavity should be aspirated with oxygen replacement.

Dermoid Cysts and Teratomata.

These tumours originate in the mediastinum, but they are dealt with in this book because the symptoms which they produce almost invariably suggest disease of the lung or of the pleural cavity. Dermoids are rare, and teratomata even more so, while only a small proportion of the tumours grow sufficiently large to give rise to clinical manifestations. At about the age of puberty they may increase rapidly in size and then displace, surround and become adherent to the adjacent structures. The earlier symptoms are due to the compression of or erosion into a bronchus, or to infection of the cystic tumour. In the former case, the cough, the sputum often tinged with blood, and the dyspnœa suggest the presence of some intrapulmonary lesion such as tuberculosis, whilst in the latter case a localised empyema is almost invariably diagnosed. The condition is seldom recognised until hairs are seen in the sputum in the pulmonary type, or the cyst is opened with the idea of draining the localised empyema, and hairs and sebaceous matter are noticed in the escaping pus. In Ogle's case,* the patient "had been ill with cough and occasional hæmoptysis for five years intermittently. The physical signs suggested empyema. His temperature was of a hectic type, and the sputum offensive, so that the diagnosis of bronchiectasis was discussed." The tumour had invaded the lung and had "caused a bronchiectasis by pressure on a bronchus, and into this it then projected, forming adhesions to its wall."

* Transactions of the Pathological Society of London, Vol. 48, 1897, page 37.

The dermoid may be a smooth-walled cyst containing masses of hair and sebaceous material only ; or it may be more complex as in Godlee's case* which had finger-like processes growing from the walls. These intracystic masses were covered by epidermis from which projected numerous long hairs.

TREATMENT.

It is possible to suggest the general principles only of the treatment of these mediastinal tumours. Whether they are producing symptoms by pressure, by erosion into a bronchus, by invasion of the lung or as a result of secondary infection, it is essential that free exposure of the interior of the cyst be obtained by removal of as much as possible of the most superficial part of the cyst wall, and of the overlying ribs and cartilages and soft tissues. If the cavity is infected, further treatment must be postponed until the acuteness of the suppurative processes has subsided. It is not possible to remove a large dermoid by dissection owing to its ramifications, and the adherence of its walls to the important vessels in the mediastinum. The best results are obtained by piecemeal removal of the pedunculated masses and cauterisation of the lining epithelium of the rest of the cavity.

The following is a brief account of the author's case of mediastinal teratoma :—

F. W., male, aged 21, was admitted into the Lewisham Infirmary in March, 1913. He had a red and tender swelling over the upper part of the left chest. This had been noticed first six weeks previously and had increased rapidly in size. The temperature was 104·8° F., and the patient was ill. An incision was made into the swelling through the 3rd space, and pus, "which was of a peculiar consistency," escaped. As there was little improvement a counter-opening was made by removing a portion of the 4th rib in the anterior axillary line. The patient's condition became worse and he was now expectorating large quantities of offensive sputum. Hairs were observed in the pus and the diagnosis of dermoid was made. The patient was transferred to University College Hospital.

The infected cavity was found to extend from the anterior axillary line on the left side to some 2 to 3 cm. to the right of the sternum, and from the left 4th intercostal space below, upwards to under both 1st costal cartilages. Adequate drainage was not obtained until a part of the left 3rd and 4th ribs and the whole of their costal cartilages, the right 2nd and 3rd costal cartilages and the intervening part of the body of the sternum had been excised. The tumour consisted of big irregular fleshy masses from which projected smaller rounded processes and hairs. The patient improved very considerably with the free drainage, but suffered such great shock from any prolonged operation under chloroform that all the subsequent

* Fowler and Godlee. "Diseases of the Lung," 1898.

operations were done under gas and oxygen. During the months July, 1913, to January, 1914, the superficial parts of the teratoma were separated from the surrounding structures and were excised. The patient was then sent away to convalesce. On his return in June it was found that the main mass of the growth had come up to the surface, replacing those parts originally removed (Fig. 80 illustrates the condition at this stage). The greater part of the teratoma was now separated from the pericardium, the aorta and other adjacent structures, and after five operations a mass measuring 13 by 8 by 7·2 cm. was excised. In November the remaining mass, measuring 7 by 5 by 4 cm. was removed. The pericardium and surrounding structures became covered by granulations, and these in turn by epithelium. There is now a cavity in his chest measuring some 2 to 3 cm. in depth, the floor of which is formed of firm scar tissue. There are two small areas less than a centimetre in diameter which continue to discharge. The patient is a strong healthy man who spends his whole day at munition work.

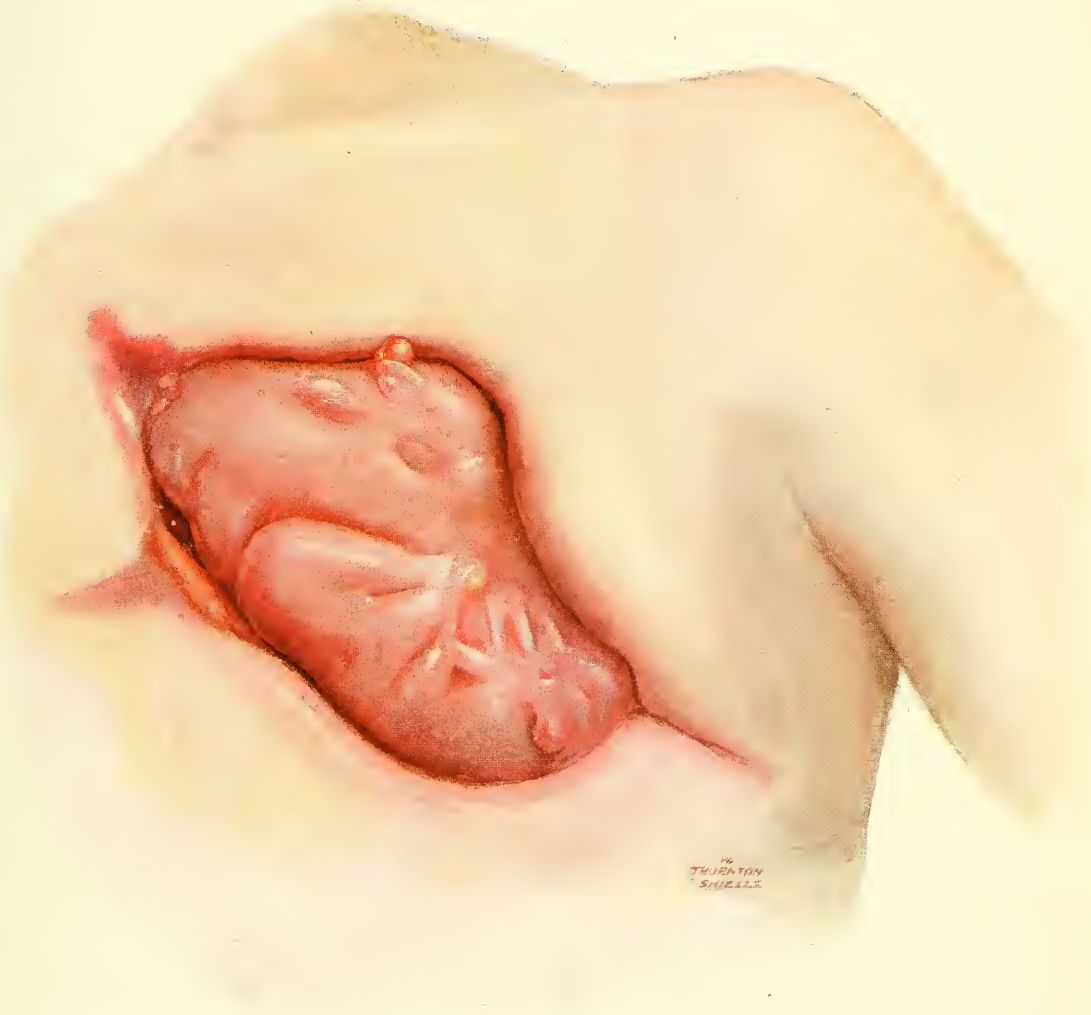

Fig. 80. Mediastinal teratoma.

CHAPTER XIII.

EMPHYSEMA.

The term "emphysema" is a comprehensive one and includes several different groups of changes in the lungs which differ from each other both in their pathological and in their clinical aspects. In this chapter it is that variety only which is known as the "large-lunged" emphysema which is under consideration. This type affects the whole of both lungs; it is associated with certain well-defined changes in the chest wall, the most striking of which are dilatation and rigidity.

In 1858 Freund first enunciated his hypothesis that in all cases of emphysema associated with a rigid and dilated thorax, the rigidity and the expansion of the chest wall were due to calcification of the costal cartilages which was the primary lesion, and that the emphysema was a secondary, and therefore a subsequent, condition. This hypothesis attracted comparatively little attention until in 1910 Freund read a paper on the subject at the Congress der Deutschen Gesellschaft für Chirurgie; on the same occasion patients were shown on whom the operation of chondrectomy, as suggested by Freund, had been done for the relief of the pulmonary symptoms. The very striking improvement in the condition of these patients aroused an interest in the subject which a reiteration of the hypothesis failed to do, and as a result of this, numerous cases of emphysema with a dilated and rigid or semi-rigid thorax have been operated on with a varying degree of success.

Freund's suggestion as to the cause of emphysema is not supported by the more recent work that has been done on the subject,* but at the same time there can be no doubt that the operation which he recommends is of very considerable value in certain cases. Our knowledge, however, on this matter is most incomplete and much work has yet to be done for the investigation of the causation of emphysema and of the treatment of it. As yet there is no clear knowledge as to the way in which the operation of chondrectomy exercises its beneficial effect on the patient, and as to whether

* The investigations of Douay, "Thorax et l'emphysème," and of the author, "The Influence of the First Costal Cartilage on Apical Tuberculosis," show that the ossification changes in the cartilages are probably due to age and muscular exertion; they are rarely found before the third decade of life in man, or before the fourth in woman. These changes tend, undoubtedly, to increase the rigidity of the chest wall, but do not predispose to dilatation.

even its action is exerted primarily on the respiratory or on the circulatory system or on both. Until this knowledge is obtained, it is therefore not possible to determine with precision what constitutes a suitable case for operative intervention or what phenomena must be regarded as contra-indications to this method of treatment. The alleviation of the distressing symptoms which has been effected in some patients, as illustrated by the case detailed below, is, however, sufficiently great to show that the operation of chondrectomy, or some modification of it, is of great value, and will, with increase in our knowledge, become more extensively employed in the future.

The patient was a man aged 40, who had suffered from emphysema for several years, and each winter was invalided by severe attacks of bronchitis. When transferred to my care in October, 1911, he had already been in the medical wards for several weeks and during this time there had been no improvement in his condition. He was cyanosed and the accessory muscles of respiration were in constant action. The paroxysms of coughing were frequent and oxygen was necessary to relieve the intense dyspnœa. Ten days after the operation of chondrectomy had been done, this patient, who previously had been unable to leave his bed owing to his shortness of breath, could run up and down two flights of stairs and breathe and talk quite easily immediately afterwards. The cough disappeared for a time and the patient no longer suffered from the sensation of a tight band constricting his chest.

Some eighteen months later there was a return of his bronchitis, and he died about two years after the operation, but up to the end he referred all his distressing symptoms to the left side and maintained that his right side was "grand."

The large-lunged emphysema is not so much a disease as it is a degeneration of the organ ; it is most probably not secondary to the dilatation of the chest wall, but is the cause of it. The striking morbid anatomy changes are the disappearance of the elastic fibres or their replacement by fibrous tissue throughout the whole of the organ, and the dilatation and distension of the alveoli and of the inter-alveolar passages. The walls bounding these spaces become atrophied and rupture, so that neighbouring alveoli coalesce. The lining epithelium undergoes degeneration. The circulation through the capillaries is greatly interfered with owing to the compression of these vessels and the obliteration of them by thrombosis and by the breaking down of the inter-alveolar walls.

The increase in the volume of the lung influences the surrounding structures. The diaphragm is depressed and there is dilatation of the chest, but it is probable that the influence exerted by the distended lung directly on the chest wall is quite a subsidiary factor in the production of the extreme degree of dilatation of the thorax ; this applies also, but to a less extent, to the downward displacement of the diaphragm. If the chest wall is

opened and air is admitted into the pleural cavity, the lung does not collapse owing to the loss of the elastic fibres ; it may even bulge slightly through the opening.

The changes in the thorax are equally striking. In an advanced case the chest is in the position of extreme inspiration. The obliquity of the ribs is decreased whilst the interval between them is increased. The sternum is pushed forwards and the obtuseness of the angle between the manubrium and the body is diminished whilst the costal angle is widened. The normal movements of respiration are absent ; instead the whole thorax is drawn upwards during inspiration and the extraordinary muscles of respiration are brought into play. The thorax as a whole is rigid. There is no doubt that when there are marked calcification changes in the cartilages, the rigidity is greater than when these changes are absent, but the calcification and perichondrial thickening are not primarily responsible for the dilatation or for the rigidity. They are an incidental condition dependent on the age and on the muscular activity of the individual.

The cardio-vascular system is greatly embarrassed by these changes in the lungs and in the walls of the thorax. The circulation is interfered with as a result of the obliteration of many of the capillaries in the inter-alveolar walls, and by the absence of the normal respiratory movements and of the normal variations in the intrathoracic pressure. The condition is further aggravated by the bronchitis, which is an almost constant complication, or by asthma, which is occasionally associated with the emphysema. Sooner or later the symptoms and signs of dilatation and hypertrophy of the right heart, and especially of the right auricle, are added to the clinical picture.

Assuming that emphysema is the primary lesion—and all the evidence indicates that this is so—it is comparatively easy to follow the stages which lead to the classical clinical picture of the barrel-shaped chest, *i.e.*, the rigid and dilated thorax. The act of inspiration is entirely an active one, but the act of expiration is dependent to a great extent on the elastic recoil of the lung, and to a less extent on the elastic recoil of the chest and on muscular action. In emphysematous patients the elasticity of the lung is at first diminished and later abolished. In order to obtain the full inspiratory complement of air, a progressive increase in the action of the inspiratory muscles becomes necessary. As time goes on these muscles, therefore, exert an increasing action on the thorax, whilst the expiratory force becomes progressively less. The result of this is a gradual expansion of the chest until it is dilated to the maximum inspiratory position. During the later stages, the extraordinary muscles come into play and respiration is carried on by the rise and fall of the chest as a whole. The loss of elasticity of the lung is thus directly responsible for the dilatation of the chest and may be regarded as the passive factor responsible for this change. There is also an active factor. The degeneration of the epithelium lining the alveoli, and the interference with the capillary circulation, together with the diminution

of the vital capacity resulting from the loss of elastic recoil, interfere with the perfect aëration of the blood and lead to a reflex stimulation of the inspiratory muscles.

The progressive contraction of the inspiratory muscles, which probably passes in the later stages into a state of tonic spasm raising and holding the ribs in the maximum inspiratory position, is in part responsible for the rigidity of the thorax ; this rigidity is dependent in part also on the secondary changes which take place in the articulations, and in the ligaments and capsules surrounding the joints. These morbid processes are capable of producing almost complete immobility, even when there is no ossification of the costal cartilages, and a much greater degree of rigidity than can result from ossification alone, even when extensive.

TREATMENT.

The operation of chrondrectomy for emphysema of the lungs consists of the removal of the first five cartilages on the right side. The effect of this is to allow of an increase in the movements both of the right and of the left side of the chest during inspiration and expiration. The respiratory movements of the two sides are not, however, synchronous. During the inspiratory phase, there is expansion of the left side of the chest, but on the right there is approximation of the cut ends of the cartilages (*i.e.*, a diminution of the side), and a sinking in of the scar tissue. During expiration, the left side of the chest diminishes in size whilst the right increases and the scar tissue is bulged outwards. It would appear at first sight that the increase in the movements of the chest wall was counteracted by the asynchronous action, but the spirometer shows that there is actually a slight augmentation of the respiratory capacity. The improvement, moreover, in the patient's general condition, in the correctly-chosen cases, and the rapid diminution of the cyanosis and dyspnœa, show that some change of a highly beneficial nature has undoubtedly been produced.

In an advanced case of emphysema, the stagnation of the air in the bronchi is very considerable, and partly on account of this and partly as a result of the obliteration of many of the capillaries, the blood has great difficulty in exchanging the CO_2 for oxygen.

Douay believes that the removal of the cartilages has a direct effect on the right auricle similar to that produced by cardiolysis in cases of adherent pericardium. It seems more probable, however, that the improvement of the right heart is secondary to a better aëration of the blood, and that this is directly due to a better ventilation of the lung. This suggestion is based on the asynchronous action of the two sides of the chest. During inspiration the air taken into the left lung is probably drawn partly from the trachea, but partly also from the right lung. This causes an increase in the negative

pressure on that side and a partial collapse of the lung and of the chest wall and sinking in of the scar. During expiration, while some of the air which is expelled from the left lung is driven out through the trachea, some of it is also driven into the right lung, causing this organ and the chest wall to expand and the scar to bulge outwards. The stagnation of the air in the bronchi is by this means abolished, and instead, a constant to and fro current is established which is greatly increased by coughing. It is probable also that the general intrathoracic circulation is at the same time considerably improved by the greater variations in intrapleural pressure.

If we assume that the above hypothesis is in the main, at any rate, correct, it is obvious that most cases of emphysema which are associated with a dilated and rigid or almost rigid chest, and in which there are signs of failure of the right heart, will be benefited by chrondrectomy. Cyanosis, dyspnœa, chronic bronchitis, and even dilatation of the right auricle do not constitute contra-indications. Acute bronchitis, a failing systemic circulation, albuminuria and acidosis are, however, strong contra-indications. The operation should not be done so long as the patient's condition responds readily to medical treatment, nor so long as a fair degree of movement of the chest wall is possible.

When operative treatment has been decided on, there are two preliminary steps which are advisable. The patient should be in bed and be treated medically at least a fortnight before the operation, so as to diminish, if possible, the bronchitis and improve the tone of the heart. A vaccine should also be given in order to reduce the virulence of the organisms in the bronchi.

Technique of Chondrectomy.

A general anæsthetic is not suitable for these patients ; the operation is quite easily done with local analgesia. At the same time as the infiltration of the tissues is carried out, the intercostal nerves should be injected with absolute alcohol. This is of very considerable advantage in the immediate after treatment, as it abolishes the greater part of the pain and enables the patient to cough and to rid himself of the bronchitic secretions.

The incision and the technique of the removal of the cartilages has already been described on pp. 215 *et seq.* When the chondrectomy is done for emphysema, the first five cartilages only need be removed. The excision of the first is the most difficult, but an essential part of the operation.

If the injection of alcohol has not succeeded in abolishing the whole of the post-operative pain and the patient refrains from coughing, there will be a retention of the secretions. This, if allowed to continue for any length

of time, is dangerous. Morphia should therefore be given and the patient induced to cough while under the influence of the drug.

Some writers on this subject recommend that, when the operation on the right side of the chest has been successful, the left side should be similarly treated. This second operation is probably not only disadvantageous, but actually dangerous unless the right side has again become completely rigid by scar tissue and regeneration from the perichondrium.

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